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CORONARY ARTERIOSCLEROSIS IN DIABETES MELLITUS*

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THERE are approximately 500,000 living diabetics in the United States at the present time, of whom more than one-half are destined to die of some form of occlusive vascular disease. While the mortality resulting from gangrene of the feet has fallen, the mortality from gangrene of the heart has risen steadily during the past few years.

The significance of arteriosclerotic (coronary) heart disease in diabetes mellitus has not been fully appreciated. During a study of the clinical and pathologic aspects of arteriosclerotic heart disease in diabetes, a complete study was made of a series of 175 diabetic patients with postmortem examinations made at the New England Deaconess Hospital during the period from 1921 to 1933.

The clinical records of each case were complete and covered periods of many years. The pathologic reports were made by Dr. Shields Warren. Careful dissections had been made of the coronary vessels. Many microscopic sections of the heart and coronary vessels were studied, utilizing a variety of tissue-staining methods. A comparable series of nondiabetic patients of the same age group, who had come to autopsy, was utilized for comparison of pathologic findings.

CASES

The series was composed of 93 patients who had presented clinical evidences of hypertension, and 82 patients who did not have hypertension. Those patients who had exhibited systolic blood pressures of 150 milligrams of mercury or more were included in the hypertensive group. Some cases were included in which the systolic blood pressure was not persistently above 150 milligrams of mercury. Since many of these patients had been subjected to surgical operations for gangrene of the extremities or other surgical complications of diabetes, variations in blood pressure were commonly encountered because of the shock attendant upon such operative procedures. On the other hand, there were some patients in the nonhypertensive group who presented evidences of cardiac failure and other conditions which, when they were observed for the first time, may have masked a previously existing hypertension.

Sex: In this series were 100 women and 75 men. The hypertensive group comprised 58 women and 35 men, while in the nonhypertensive group there were 42 women and 40 men. In the last series of 83 diabetic patients who had come to autopsy between 1929 and 1930, this same relationship of females to males obtained. In the nonhypertensive group the sex distribution was about equal. It is worthy of comment that in the hypertensive group the females greatly outnumbered the males.

Age: The average age at time of death for the patients of the hypertensive group was 62.4 years; for the nonhypertensive group the average age was 54.4 years. A number of relatively youthful patients in the non-hypertensive group appreciably lowered the average age. Possible confusion in evaluating this factor was avoided by also comparing the extent of coronary disease in each group of patients above 40 years of age. The average duration of diabetes in the hypertensive group was 7.9 years; in the nonhypertensive group it was 6.0 years. In the complete series of autopsied patients there were 46 persons whose diabetes had been present for 10 years or more. Twenty-four of these patients were women and 22 were men. The youngest person in the group of those who had had diabetes for 10 years or more was 43 years of age, with diabetes for 15.1 years, while the oldest was 83 years of age, with diabetes of 33.7 years' duration.

Obesity: Obesity was a common finding in both groups. Obesity was present in 87.2 per cent of the patients in the hypertensive group and in 74.7 per cent of those in the nonhypertensive group. The degree of obesity ranged as high as 90 per cent overweight.

Incidental Factors: Possible predisposing factors, such as race, occupation, infection, heredity and endocrine dysfunction, were considered, but none appeared to play any dominant rôle in this series. Rheumatic heart disease was present in 6 patients (Case Nos. 11,257; 11,692; 10,192; 6,380; 6,572; 5,983). Nine patients had positive serologic evidence of syphilis, but

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none showed any evidence of syphilitic heart disease at autopsy. One patient had persistent auricular fibrillation, secondary to hyperthyroidism. The pathologic changes found in the hearts at time of autopsy were predominantly due to arteriosclerosis.

Control Series: The control series comprised a total of 170 consecutive autopsied nondiabetic patients, all of whom were over 40 years of age and who died during 1929 and 1930. The average age at time of death was 64 years. There were 87 females and 83 males. The changes in the coronary vessels were graded "0" to "4", depending upon the extent of the arteriosclerotic changes found. "Zero" denoted no atheromatous changes, while "4" indicated extreme coronary atherosclerosis with obliteration of the lumen, calcification and thrombosis.

Data: Table 1 shows the distribution of coronary disease among the hypertensive and nonhypertensive diabetic groups in contrast to the control series. Slight atheromatous changes occurred with equal frequency, but striking differences are noted in the occurrence of more advanced vascular changes. Severe degrees of obliterative coronary disease occurred in 60 per cent of the hypertensive group and in only 22 per cent of the nonhypertensive group of the same age. The total incidence of severe coronary disease in the 175 diabetic patients was 42 per cent; fatal coronary thrombosis occurred in 18 per cent. Deducting all patients under 40 years of age reveals an incidence of 46.7 per cent of severe coronary disease and 19.5 per cent of terminal coronary thrombosis.

In the control series of 170 nondiabetic patients, severe coronary disease was present in only 13 per cent, despite the fact that no patients under 40 years of age were included in this group and despite the fact that the average age at time of death was 61.9 years. Coronary thrombosis was present in only 4 cases (2 per cent).

Coronary heart disease was a cause of death in 36 patients (39 per cent) in the hypertensive group. All but one of these patients died primarily as a result of insufficiency of the coronary circulation, secondary to obliterative changes in the coronary arteries. This is in direct contrast to the higher incidence of myocardial insufficiency found in nondiabetic patients with hypertension. In the entire diabetic series only one patient (Case No. 4,304) died as a result of myocardial insufficiency, secondary to hypertension, without advanced obliterative changes in the coronary vessels. In the nonhypertensive group, coronary heart disease caused death in 12 patients (15 per cent). Three of these patients (Case Nos. 6,798; 5,248; 9,433) died of congestive heart failure following acute coronary thrombosis.

Myocardial degenerative changes occurred in 78 per cent of the hypertensive group of patients and in 43 per cent of the nonhypertensive group. By eliminating from consideration all patients under 40 years of age in the nonhypertensive group, this incidence would be raised to 52 per cent. The control series revealed an incidence of myocardial degeneration of 79 per cent. No striking differences were noted in the type or degree of arteriolar sclerosis in the two

TABLE 1

CORONARY ARTERIOSCLEROSIS IN 175 DIABETICS AT AUTOPSY

93 Cases with Hypertension

Cases	Grade of Sclerosis				
	0	1	2	3	4
Number	7	15	15	31	25
Per cent	8	16	16	33	27
Age	61.1	57.6	65.7	65.9	61.7
Duration D. M.	3.2	6.9	6.8	11.3	11.6
Heart weight	360.0	419.0	450.0	393.0	412.0

82 Cases without Hypertension

Cases	Grade of Sclerosis				
	0	1	2	3	4
Number	36	13	15	11	7
Per cent	44	16	18	13	9
Age	42.9	56.8	58.0	59.4	54.7
Duration D. M.	3.5	3.7	7.5	7.2	8.1
Heart weight	294.0	335.4	286.0	396.0	376.0

Non-Diabetic Control Series

Cases	Grade of Sclerosis				
	0	1	2	3	4
Number	62	48	37	19	4
Per cent	37	28	22	11	2
Age	57.7	63.9	56.4	68.5	64.8
Heart weight	297	290	353	369	452

Key to Table

0 No Coronary Sclerosis
1 Slight Coronary Sclerosis
2 Moderate Coronary Sclerosis
3 Severe Coronary Sclerosis with Obliteration of Lumen
4 Severe Coronary Thrombosis Resulting in Death

Heart Weight Given in Grams.

groups of corresponding ages. The histologic changes associated with senescence predominated in each group.

Valvular sclerosis and atheromatosis occurred in 25 patients in the hypertensive group and in 7 patients in the nonhypertensive group.

The relationship of the weight of the heart to the degree of coronary sclerosis is indicated in table 1. In the hypertensive group the average weight of the heart was 407 grams, while in the nonhypertensive group the average weight was 339 grams. If the patients under 40 years of age in the nonhypertensive group were excluded, it would raise the average heart weight to 350 grams. In the hypertensive group the average heart weight for groups 3 and 4 was somewhat less than in the preceding groups. This was not true for the nonhypertensive diabetic patients or for the nondiabetic control groups, however. The fact that hypertrophy of the heart did not occur in a large number of patients with hypertension may be seen from an inspection of table 1. Indeed, in a large number of cases the term "atrophy" might be used in describing the size of the heart, even though many of these patients exhibited signs of cardiac disease. The average heart weight for the control group was 352 grams.

We have arbitrarily chosen 350 grams as the upper limit of normal. Some authors have considered 450-500 grams to represent the upper limit of normal weight of the heart. Consequently, it will be found that many more hearts have been included in the hypertrophic group than would be the case if we adopted the higher figures.

Gross acute infarction of the myocardium was observed in 25 cases in the hypertensive group, while old infarcts were found in 21 cases. An unusually high incidence (4 cases) of rupture of the myocardium was found. Aneurysm of the ventricle occurred in one case. In the nonhypertensive group, gross acute infarction was present in 7 instances, while healed infarcts were found in 6 cases. Aneurysm of the ventricle was also observed in 1 case in this group.

The histopathologic changes were identical in type in both the hypertensive and nonhypertensive groups. They were essentially intimal changes of the atheromatous and fibrous proliferative types. The atheromatous changes predominated. These lesions were more commonly observed in the larger vessels of the coronary tree. Both the right and left coronary arteries and their larger branches were affected by the process. Coronary thrombosis occurred in the right side of the heart in only 1 case (Case No. 177). The pathologic process was not uniformly diffuse, but occurred as a patchy lesion. Occlusive vascular changes in the coronary arteries were frequently associated with localized areas of myocardial atrophy which, despite some degree of adjacent compensatory hypertrophy, did not increase the weight of the heart materially. Terminal thrombosis occurring at the site of an atherosclerotic plaque produced sudden death in 20 cases; 15 of these patients were in the hypertensive group.

Warren[1] found marked glycogenic infiltration of the heart muscle in many of these cases. It was particularly striking in the heart muscle fibres in the margins of the infarcts. Abnormally large storage of glycogen was found particularly in hearts subjected to fixation within thirty minutes after death. There was but little difference in the amount of glycogen stored in the hearts of diabetic patients who had received insulin and in the hearts of the control patients. In the hearts of diabetic patients not treated with insulin there was a high proportion showing heavy deposits of glycogen.

Symptomatology: When cardiac symptoms were present in this series of patients they were predominantly those associated with coronary heart disease. The most characteristic symptom of coronary disease in the patients comprising this series was the gradual onset of substernal pain of a relatively mild type, first induced by effort and rarely so severe as to be incapacitating. Dyspnea of cardiac origin was not a common symptom. Nocturnal dyspnea or paroxysmal pulmonary edema rarely occurred. Congestive heart failure was infrequent; when it occurred it usually followed coronary thrombosis. Angina pectoris had been present in but 16 of the 32 patients with coronary thrombosis. The duration of symptoms was often only a few months before terminal coronary occlusion. In 8 of the cases of coronary thrombosis there was no history of pain. In 2 cases coronary thrombosis was signalized by the sudden onset of auricular fibrillation. In 2 cases, coronary thrombosis occurred in patients who had insulin hypoglycemia. It was not possible to show that the hypoglycemia preceded the coronary thrombosis.

INCIDENCE OF CORONARY THROMBOSIS

Such a high incidence of coronary thrombosis merits special attention. Of the 32 fatal cases comprising an incidence of 18 per cent of the 175 cases, 27 per cent occurred in the hypertensive group and 9 per cent in the nonhypertensive group. In the control group coronary thrombosis occurred in only 2 per cent.

Sex: Of the 32 patients with coronary thrombosis there were 18 women and 14 men. The preponderance of males over females in other reported series of autopsy studies on nondiabetic patients with coronary thrombosis was not found in this study.

Age: The average age at time of death was 61.7 years for the patients in the hypertensive

group and 54.7 years for those in the nonhypertensive group. The youngest patient (Case No. 1,794) was 33.5 years of age and the oldest (Case No. 5,480) was 82.7 years of age. There were 8 patients, 25 per cent of this group, who were 70 years of age or older. The average duration of diabetes for this group was 17.4 years, indicating a relatively mild type of diabetes. Such a high incidence of coronary thrombosis in this age group would tend to show a more significant relationship of the duration of diabetes mellitus to coronary sclerosis than of age or the severity of the diabetes.

Obesity: Obesity maintained about the same relationship to patients with coronary thrombosis as obtained for the entire group. Eighty-eight per cent of the hypertensive patients had been obese, as compared with 71 per cent of those in the nonhypertensive group.

Duration of Diabetes Mellitus: The average duration of diabetes in the group of hypertensive patients with coronary thrombosis was 11.6 years; the duration for nonhypertensive patients was 8.1 per cent years. Among the 8 cases in which coronary thrombosis occurred in patients past 70 years of age, the average duration of diabetes was 17.4 years. The majority of these patients had a mild type of diabetes, despite the fact that diffuse arteriosclerosis was found at postmortem examination in addition to coronary arterial disease. Coronary sclerosis, as with arteriosclerosis elsewhere, bore a closer relationship to the duration of the diabetes than it did to the severity of the diabetes or to the age of the patient.

DISCUSSION

In this series of 175 autopsied diabetic patients, arteriosclerosis was the cause of death in 116 (66 per cent). Forty-two per cent of the 175 patients died of severe coronary heart disease. Excluding all patients under 40 years of age, the incidence of severe coronary disease would be elevated to 47 per cent. Among the 175 patients there were 32 deaths attributable to acute coronary thrombosis, an incidence of 18 per cent. Deducting the number of patients under 40 years of age would raise this figure to 20 per cent. In 4 patients, rupture of the myocardium occurred at the site of infarction and in 2 instances aneurysm of the ventricle was found. Striking differences are shown when these figures are compared with those obtained from a study of the control series of 170 patients. Severe coronary disease (groups 3 and 4) occurred in only 13 per cent of the control patients, despite the fact that no patients under 40 years of age were selected and even though the average age for this group was 61.9 years. Coronary thrombosis occurred in only 4 patients (2 per cent). No cases of ruptured myocardium or aneurysm of the ventricle were encountered.

Many observers, from Seegen in 1870 to Naunyn in 1906,[2, 3, 4, 5, 6, 7, 8] have noted the frequent association of coronary disease and diabetes mellitus. Since the advent of insulin the common association of diabetes and coronary heart disease has been emphasized by Root and Warren[9] and Nathanson.[10] The latter concluded that coronary disease is six and one-half times as common in the diabetic individual above 50 years of age as it is in the nondiabetic patient of the same age group.

Wilder,[11] Blotner,[12] Rabinowitch[13] and, more recently, Enklewitz[14] have shown the great frequency of coronary thrombosis in the diabetic. In 10,000 patients with diabetes, Root[15] found that angina pectoris occurred in 410 cases.

On the other hand, diabetes is frequently found to be present in patients suffering from coronary thrombosis. Levine[16] found glycosuria or a previous history of diabetes in 23.7 per cent of 145 patients with coronary thrombosis.

Compared with other general autopsy series, the incidence of coronary thrombosis in the diabetic group of patients is striking. Benson and Hunter,[17] in a study of 1,750 autopsies performed in the coroner's service at Portland, Oregon, found coronary thrombosis in 4.1 per cent. One would expect a relatively high incidence of coronary thrombosis in a series of this origin. Ophüls[18] found only 18 cases of recent myocardial infarction in 3,000 nondiabetic autopsies. Coronary thrombosis was present in only 2 per cent of the cases in our nondiabetic control series. Meakins and Eakin[19] found only 62 cases of coronary thrombosis in 6,548 autopsied patients covering a period of 35 years; this series revealed a ratio of deaths from coronary thrombosis of 1:105.6 or 0.95 per cent.

The interrelationship of hypertension, hyperglycemia, obesity and arteriosclerosis has been emphasized by Herrick,[20] Kramer,[21] Musser and Wright[22] and others. The etiologic importance of obesity and diabetes is well appreciated. The exact relationship of obesity to hypertension is not definitely known. One-half of our patients had diabetes mellitus, obesity and a relatively high incidence of arteriosclerosis, yet hypertension was not present. It is true, however, that reduction of body weight in many patients with hypertension induces a favorable influence upon the blood pressure.

DURATION OF DIABETES MELLITUS

As indicated in table 1, the degree of coronary sclerosis, with occasional exceptions, bore a closer relationship to the duration of diabetes than it did to the age of the patient or the severity of the disease. The inclusion of 13 patients under 40 years of age appreciably lowered the average age level for the patients of the non-

hypertensive group. The average age of the patients with coronary thrombosis was appreciably lower than the age level of the patients showing a moderate degree of coronary sclerosis. This correlation of the existence of coronary sclerosis with the duration of diabetes parallels that found previously in diabetic patients with a duration of diabetes of five years or more. The average age in groups 1, 2 and 3 of coronary disease remained about the same. A lower average age was found in group 4 in both the hypertensive and nonhypertensive groups of patients (table 1). There is a steady progression of the severity of coronary arteriosclerosis with a progression of the duration of diabetes mellitus, despite the fact that there are no great changes in the average ages for the various groups. In both the hypertensive and nonhypertensive groups no coronary sclerosis was found in patients with an average duration of diabetes of less than five years.

In patients with diabetes of short duration there is no more arteriosclerosis than in nondiabetic patients. The excessive arteriosclerosis is found in diabetes of long duration. This correlation of the duration of diabetes mellitus to aortic atherosclerosis was previously reported by Root and Sharkey.[23] The effect of the duration of diabetes upon the degree of arteriosclerosis is likewise demonstrated in the extremes of the age groups. In our series there were 2 patients with severe coronary disease under 40 years of age. Of these 1 died of coronary thrombosis. Root and Graybiel[24] reported 2 cases of angina pectoris in patients between 20 and 30 years of age and 4 between 30 and 40 years of age. Three of these patients, who had had diabetes for 5.6 to 8 years, died suddenly; in two instances postmortem examination revealed acute coronary thrombosis.

Anderson[25] reported death of a 33 year old patient with cardiac decompensation secondary to coronary heart disease, who had had diabetes for 14 years. Klingenberg[26] reported two similar occurrences in patients between the ages of 20 and 30 years; in one an area of healed myocardial infarction was found at autopsy. Cullinan and Graham[27] reported the death with coronary occlusion of a 27 year old patient who had had diabetes for 8 years.

In the other extreme of life we find that 25 per cent of our patients with coronary thrombosis were 70 years of age or over. The average duration of diabetes for this group was 17.4 years.

Eggleston[28] states that coronary thrombosis is uncommon in patients over 70 years of age. Willius[29] found only one instance of coronary thrombosis among 700 patients over 75 years of age; he attributes this to the fact that the majority of patients who reach advanced ages do so because they possess "a sturdy coronary tree". Willius stated, "The extremely small incidence of coronary thrombosis (0.3 per cent) in this series substantiates previous observations that its occurrence is most common between the fiftieth and seventieth years of life." In his series, coronary disease was present in 44.7 per cent. In our series of diabetic patients the incidence of severe coronary disease was 42 per cent, despite the fact that the average age of the patients of our series was approximately 13 years less than that in Willius's series.

Although diabetes develops most frequently at 51 years of age, angina pectoris occurs most frequently in diabetic patients during the decade in which patients who have had diabetes for 10 years or more are most frequently encountered, namely, the seventh. White's[30] series reveals that the greatest frequency of onset of angina pectoris among nondiabetic patients occurs during the sixth decade. The later development of angina pectoris in diabetic patients appears to depend upon the fact that the exposure to diabetes of several years' duration is an important factor in the etiology. The frequency of angina pectoris trebles during the second decade of diabetes.

HYPERTENSION

The almost equal distribution of patients with hypertension and those without hypertension in our series provided an opportunity to compare the pathologic changes observed in the two groups. In general a greater frequency of hypertension in diabetic patients than in nondiabetics had been reported by many authors, including Major,[31] Bell and Clawson,[32] Peterson,[33] Root and Graybiel,[24] Koopman,[34] Rabinowitch et al.[35] and Nathanson.[36]

The type of hypertension occurring in our diabetic patients corresponded to the so-called benign type. Few of the patients had symptoms attributable to the hypertension per se. There was a notable absence of headache, vasomotor disturbances, central nervous system symptoms or epistaxis. On the contrary, Weiss[37] found only 11.8 per cent of 1090 ambulatory nondiabetic patients with hypertension to be free of symptoms ordinarily associated with this disease. So-called malignant hypertension was not observed in any of our patients and none of the pathologic changes ordinarily associated with malignant hypertension were present. While the duration of hypertension in our patients was difficult to estimate, it developed after the onset of the diabetes in many cases in which diabetes had existed for many years. While it is sometimes stated that diabetes is commonly found in persons with hypertension, the autopsy records on patients with hypertension do not confirm this. Bell and Clawson[32] found diabetes to be present in only 5.5 per cent of the patients in their series. Murphy et

al.[38] in their series of 375 autopsied patients with hypertension found a surprisingly low incidence (14 cases) of diabetes.*

The effect exerted by hypertension can be estimated by a comparison of the pathologic changes in the diabetic and nondiabetic series. The nature of the pathologic changes was approximately the same in diabetics with or without hypertension, though the incidence of severe coronary atherosclerosis was greater in patients with hypertension. The average weight of the heart for the hypertensive group was 391 grams and for the nonhypertensive group 335 grams. The relative absence of any marked hypertrophy of the heart, even in patients with hypertension, is significant. This is in direct contrast to the average weight of the heart in the nondiabetic hypertensive patients. The more extensive pathologic changes in the hearts of diabetic patients with marked obstruction of the coronary circulation probably accounts for the lack of hypertrophy.

Congestive heart failure occurred but once in the diabetic group except as a result of coronary thrombosis. Congestive heart failure on the contrary was the chief cause of death in the hypertensive nondiabetics in Bell and Clawson's[32] series; it occurred in 44.5 per cent. The weight of the heart of these patients varied from 448 to 615 grams. Murphy et al.[38] in their series of 375 hypertensive patients found only 18 per cent of the hearts to be of normal weight; the remainder varied from 400 to 1000 grams. Absence of marked cardiac hypertrophy and of congestive failure, even in the presence of hypertension, characterized the diabetic series.

Coronary disease as a cause of death was far more frequent in the diabetic than in the nondiabetic series. Only 2 per cent of the nondiabetics had coronary occlusion, whereas among the diabetics it occurred in 9 per cent of those without hypertension and in 27 per cent of those with hypertension.

DIABETES AS A CAUSE OF ARTERIOSCLEROSIS

Whether arteriosclerosis represents cause or effect of hypertension is not definitely proved, but the consensus at the present time (Bell and Clawson,[32] Fishberg,[39] Moschcowitz,[40] Musser,[41] Rabinowitch,[35] Allbut,[42] Mosenthal,[43] Yater,[44] Rice,[45] Goodridge,[46] White[47] and others) is that arteriosclerosis in many cases is an effect, rather than a cause, of hypertension. In this series coronary atherosclerosis stood in a much closer relationship to the duration of diabetes than to the age of the patient. The frequency of coronary thrombosis at the extremes of age, as reported by ourselves and other authors, is characteristic of diabetes. The sex relationship to severe coronary disease in our series is also distinctive of diabetes. Eighteen of our 32 patients with acute coronary thrombosis were women. The higher incidence of coronary thrombosis in females is in direct contrast to other published reports on nondiabetic patients, such as Willius's[66] recent report in which the males outnumbered the females in the ratio of seven to one. Sprague and White[48] state that 90 per cent of their patients under 50 years of age with coronary heart disease were men.

Sprague and White[48] agree that diabetes favors the early appearance of coronary atherosclerosis. Warren[49] has emphasized the selectivity of the vascular pathologic lesions in diabetes. A characteristic type of intimal atherosclerosis is associated with diabetes, involving principally the muscular arteries, such as those of the heart and legs. This type of arteriosclerosis may be contrasted with that form which is seen in the average senile patient, involving chiefly the cerebral and renal vessels.

Among our 175 autopsied patients cerebral arteriosclerosis was no more frequently a cause of death than in nondiabetic patients. In only 1 case in our series could death be ascribed to nephrosclerosis. Further confirmation of the selectivity of the vascular pathologic lesions associated with diabetes is advanced by Waite and Beetham[50] who found retinal sclerosis to be as frequent in nondiabetic as in diabetic patients.

If it were true that diabetes mellitus occurs secondary to a generalized degenerative arteriosclerosis, especially localized in the pancreatic vessels, and that diabetes bears no causal relationship to arteriosclerosis, we would expect an increasing incidence of diabetes with age. Joslin[51] has shown that the maximum susceptibility to the development of diabetes occurs at 51 years of age for men and 55 years of age for women. In the later decades, when arteriosclerosis increases, diabetes decreases. Approximately one-third of the cases of diabetes develop during the first four decades. If arteriosclerosis is the cause of diabetes, one would expect some direct relationship between the degree of arteriosclerosis and the severity of the disease; such is not the case. Patients developing diabetes after the age of 60 almost invariably exhibit a mild type of the disease. In our autopsied series of 175 patients there were 46 with a duration of diabetes of 10 years or more. Arteriosclerosis was the cause of death in 66 per cent of these patients; coronary disease produced death in 52 per cent. The youngest patient was a man, 43 years of age (Case No. 3,286), who had had diabetes for 15.1 years. This patient died of coronary heart disease; he showed evidence of retinal sclerosis with hemorrhages, had gangrene of his toes, and, at autopsy, revealed marked aortic atherosclerosis with calcification. The oldest patient, a woman, 83 years

*Personal communication.

of age (Case No. 5,480) had had diabetes for 33.7 years. This patient died of coronary thrombosis; she had radial arteriosclerosis and gangrene of the legs and, at autopsy, marked aortic atherosclerosis with calcification and ulceration was found. These 2 patients represent the extremes of life in this particular group; each died of coronary heart disease and showed diffuse arteriosclerosis. The mildness of the diabetes in these two cases is indicated by its long duration. To quote Woodyatt[52]: "No really severe diabetes would have continued for so long as this."

Pathologic evidence is likewise against the concept of arteriosclerosis as a cause of diabetes. There was no more arteriosclerosis in patients who had had diabetes for a short interval than there was among nondiabetic patients. Warren[53] found as much pancreatic arteriosclerosis in a control series of nondiabetic patients as he did in diabetic patients. In his series of 359 diabetic cases in which it was possible to study the pancreas, no pathologic changes could be demonstrated in 69 cases (19 per cent). Furthermore, a high incidence of pancreatic arteriolar sclerosis is reported in hypertensive patients without diabetes.

Fahr[54] found that the small arteries of the pancreas are usually sclerotic in cases of hypertension. Bell and Clawson[32] expressed the impression that pancreatic arteriolar sclerosis was as common in their series as in Fishberg's series, yet diabetes had been present in only 5.5 per cent of patients over 50 years of age. Murphy et al.[38] found arteriolar lesions in the pancreas similar to those found in the kidneys in patients with hypertension.

The mechanism by which diabetes induces the development of atherosclerosis is still under discussion. The evidence suggests that cholesterol metabolism is a primary factor. The work of Virchow[55] and Aschoff[56] suggested that the development of atherosclerosis was related to disturbances of cholesterol metabolism. More recently other authors (Minovici and Vangheloviei,[57] Labbé and Heitz,[58] Lehnherr,[59] Gibbs, Buckner and Bloor,[60] Schönheimer,[61] Warren,[62] Rabinowitch[35] and others) have provided further evidence of an etiologic association between cholesterolemia and the premature and excessive production of atherosclerosis in the diabetic patient. The most convincing recent work on this subject is that of Leary,[63] who has reproduced typical coronary atherosclerosis with occlusion in animals by feeding cholesterol. As noted in the first report of autopsies from the Deaconess Hospital, the frequency and advanced character of coronary and general atherosclerosis is most clearly explained by the disturbances in lipid metabolism. Other factors, such as heredity, stresses and endocrine influences, may play a part, but the abnormal cholesterol metabolism appears to be the most important cause.

TREATMENT

The usual cardiac drugs have proved to be of limited value in the treatment of coronary heart disease occurring in patients with diabetes mellitus. Drugs with vasodilator action, such as nitroglycerin, have been used, but most patients place more reliance upon restriction of activity and periods of rest. Surgical measures have not been recommended because the pain is rarely of a severe degree and the extent of the coronary sclerosis is usually quite advanced.

Insulin administration in the diabetic patient with coronary heart disease should be accurately gauged on the basis of frequent blood sugar and urine analyses. It is unfortunate that reports of isolated instances of the development of angina pectoris or coronary thrombosis following insulin-hypoglycemia have led to considerable prejudice against the use of insulin. The carefully controlled administration of insulin in cases of coronary heart disease in diabetic patients is of proved value. A marked loss in carbohydrate tolerance occurs in some cases of coronary thrombosis, particularly if congestive heart failure is present. Insulin therapy is of distinct value in such cases. Hepburn and Latchford[64] have shown that insulin therapy increased the average sugar consumption of the heart muscle from the normal rate of 0.87 milligrams per hour to 3.06 milligrams per hour. Lymburner[65] has described a patient who had experienced a severe attack of angina pectoris, possibly following coronary occlusion, and who was given 250 units of insulin within twelve hours with excellent results. The recovery of many diabetic patients following coronary occlusion provides evidence that uncontrolled diabetes in such patients should be regulated by the judicious use of insulin.

SUMMARY AND CONCLUSIONS

1. Among 175 diabetic patients who were subjected to postmortem examination, coronary arteriosclerosis was the most common cardiac lesion and was present in some degree in 132 cases.

2. The clinical symptoms of cardiac disease, when present, were those of coronary sclerosis. Angina pectoris was always associated with severe coronary disease.

3. The degree of coronary sclerosis bore a more definite relationship to the duration of the diabetes than it did to the age of the individual or the severity of the disease. This relationship held true for both hypertensive and nonhypertensive diabetic patients.

4. A much higher incidence of severe cor-

onary disease was encountered in those cases in which diabetes and hypertension co-existed.

5. The incidence of coronary thrombosis in the diabetic patients was much higher than it was in a comparable control series of nondiabetic patients or in other reported series of general autopsy material. Coronary thrombosis occurred with approximately four times greater frequency in the diabetic patients with hypertension than it did in diabetic patients without hypertension.

6. Pathologic changes of an atherosclerotic nature were found most consistently in the main coronary arteries and their larger branches.

7. Cardiac hypertrophy was not encountered so frequently as one would expect in diabetic patients with hypertension.

8. A statistical analysis of the incidence of diabetes, combined with a study of pathologic changes found at autopsy, reveals no evidence that arteriosclerosis is a cause of diabetes.

9. Prolonged duration of diabetes mellitus exerts a marked etiologic influence on the development of atherosclerosis.

10. The occurrence of coronary thrombosis in diabetic patients is not a contraindication to the employment of insulin therapy.

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CONGENITAL DISLOCATION OF THE HIP*

An End-Result Study with Suggestions for Improved Treatment

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THE purpose of this paper is to present a brief survey of the experiences of the orthopedic staff of the Massachusetts General Hospital with congenital dislocation of the hip.

All of the cases treated during a ten year period (1923-1933) have been reviewed and classified according to the method of treatment employed. There were 58 cases in all, representing a total of 73 dislocated hips. Of these, only 46 patients, comprising 55 hips, could be followed to the end-result.

The treatment of these cases was shared by different members of the orthopedic staff and consisted in closed reductions, open reductions, open reductions with shelves, and shelf operations alone, depending upon the nature of the case. Traction of the Hoke type or by Kirschner wire has been used both before and after reduction in about one-half of the open reduction cases. No Lorenz bifurcation operations or Shanz osteotomies have been performed. The method of closed reduction employed was that which was advocated by Denucé.[1] The open reductions were all performed through the Smith-Petersen approach to the hip. In those cases where shelves were employed, various kinds were constructed, but chiefly the turning down of a bone flap from the blade of the ilium plus a bone wedge to retain the flap in its position. In several instances, a "trap door" type of bone shelf was used, raising the bone ledge from below upward. Also, in a few cases, full thickness grafts were taken from the tibia and driven at right angles into slots in the ilium, thus producing a roof over the femoral head.

The age factor has always been considered of paramount importance in congenital dislocation of the hip and cases are, therefore, frequently separated into different age groups, so far as treatment is concerned.

Dickson,[2] for instance, speaks of three groups, in which "Group I" comprises patients up to 4 years of age; "Group II" patients from 4 years to 9 years of age; "Group III" patients over 9 years of age. In Group I (up to 4 years) he recommends closed reduction. In Group II (4 years to 9 years) open reduction is advised. In Group III (over 9 years) shelf operations are the procedure of choice. Other writers suggest different age groups.

Galloway[3] places the age period from 2 to 3 years as the ideal age for open operation.

Kidner[4] believes that even in very young children open operation is preferable to the closed method, unless reduction by the closed method can be easily obtained with a minimum amount of force and with a stable hip immediately following manipulation.

Farrell and Howorth[5] state that "closed reduction" is the operation of choice in infants younger than 1 year. Between the ages of 1 and 3 years, they suggest that a single gentle manipulation first be attempted; if not successful, then open reduction. They believe that shelf stabilization, without reduction, is usually preferable in children over 6 years of age.

Gill[6] always attempts closed reduction in children under 5 years of age, unless the joint is abnormally rigid or the x-ray shows too much obliteration of the acetabulum. Over 5 years of age, he advises the open operation and always combines it with a shelf operation.

At the Massachusetts General Hospital, no arbitrary age limits have been used in determining the type of operation to be performed. As a rule, closed reduction has been reserved for children under 5 years of age unless special contraindications were apparent.

Open reductions have been performed in children over 5 and in younger children where the closed method had failed.

In the series of open reductions, a shelf operation was added only in those cases that exhibited an incompetent acetabulum.

"Shelf operations alone" were performed in adults, and in those children whose hips, obviously, could not be reduced without great trauma. In such instances, the femoral head was allowed to remain in the false acetabulum.

In the series of cases to be discussed here, the ages of the patients were as follows:

Under 6 years of age	16
6 years to 15 years	18
15 years to 30 years	11
Over 30 years	2

Of the 55 dislocated hips with end-results, 16 were treated by closed reduction, 15 by open reduction, 8 by open reduction plus a shelf, and 16 by the shelf operation alone.

Following closed reduction, it has been the custom at the Massachusetts General Hospital to immobilize the hip in its most secure position of reduction for a period of about six months and then gradually, in successive stages, to allow the leg to assume its normal anatomic position, the entire period of plaster retention not exceeding nine to ten months.

In other orthopedic clinics (A. B. Gill), the

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reduced hip is fixed in a plaster cast for only four months, in the belief that if the head of the femur will not remain in the acetabulum after four months in plaster, the probabilities are that it will not do so even after ten or twelve months in plaster. Should the hip redislocate after this period of four months' fixation, an open reduction is definitely indicated.

For the postoperative treatment of open reductions Farrell and Howorth[5] apply a plaster-of-Paris hip spica for two and one-half to three months. Active motion is begun promptly upon the removal of the plaster but weight-bearing is usually delayed from one to three weeks. They believe that mobilization of the joint should be begun at the earliest safe moment but that weight-bearing should be delayed until the extremity has recovered sufficient function.

Cole[7] makes the period of fixation in plaster for these hips two to four months; Kidner[4] three months.

The author agrees with these writers that the period of fixation in plaster should be much shorter after open reduction than after the closed method, but would make no arbitrary time limits, preferring to regulate the immobilization period according to the needs of the individual case. Some openly reduced hips will require no more than two months of immobilization, while others, because of anatomic changes about the head and neck, may require a much longer time.

Following plaster-of-Paris fixation, the early use of active motion and leg exercises under proper supervision is very important, but unfortunately often neglected in large hospital clinics.

END-RESULTS

It is very difficult to estimate accurately end-results in congenital dislocation of the hip. Since the mechanics and structure of the reduced hips change as time goes on, there is no one period that can be selected for a definite end-result.

What may be taken for an end-result at two years after reduction, may be entirely different ten years postoperatively. Traumatic arthritis, obesity, occupation, economic demands, and so forth all have their effects upon the susceptible joint, and what might be considered a good early operative result, often, in the course of time, becomes a poor end-result.

Then, too, what may be considered a satisfactory or good result by one surgeon may be considered a poor result by another. There is no standard measuring stick that has been universally adopted, and individual statistics are, therefore, difficult of interpretation.

The evaluation of results in this series of cases is entirely a personal one and must be looked upon as relative.

In general, the result termed "Excellent" in this paper signifies a perfect clinical result, without a limp, Trendelenburg sign, disability, loss of motion, shortening or atrophy, and with an x-ray in which the changes shown are insignificant. "Good" means an excellent clinical result without disability but with a slight limp, Trendelenburg sign, slight limitation of motion and significant x-ray changes. "Fair" means definite restriction of motion in more than one direction with some disability, but no marked pain. "Poor" means only a small range of motion with marked limp, shortening, considerable disability and pain.

Closed Reductions

Of the closed reductions, of which there were 16, 10 hips or 62 per cent showed "excellent" end-results; 4 hips or 25 per cent were classified as "good" results; and 2 hips or 12.5 per cent showed a "fair" result.

Open Reductions

Of the open reductions, of which there were 15, only 1 hip or 7 per cent was rated "excellent"; 3 hips or 20 per cent rated as "good"; 5 hips or 33 per cent rated as "fair"; 4 hips or 27 per cent rated as "poor"; and 2 hips or 13 per cent resulted in bony ankylosis. These last 2 hips which ankylosed were in individuals of 17 years and 39 years of age, and in both cases a stripping of all of the attachments from the neck and trochanter of the femurs was done preliminary to heavy traction and open reduction. Of the 4 hips rated "poor", 1 case re-dislocated and the patient refused further treatment; the second case had all of the structures about the neck and trochanter released and heavy traction employed prior to an attempt at open reduction. In the remaining 2 hips no definite cause for the poor results could be ascertained in view of the fact that the patients returned to the clinic only once, and x-rays and sufficient data were lacking.

Open Reductions Plus a Shelf

Of the hips which were treated by "open reduction plus a shelf," of which there were 8, no single case has been recorded as an "excellent" end-result. Two hips or 25 per cent showed "good" end-results; 1 hip or 12.5 per cent showed a "fair" result; 2 hips or 25 per cent were rated as "poor"; and 3 hips or 37.5 per cent resulted in bony ankylosis. Of the 2 cases reported as "poor", one had an open reduction which was difficult and a skid had to be used to secure reduction. A "trap-door" type of shelf was also employed, and later a subtrochanteric osteotomy was done to correct the marked adduction deformity. All of these procedures were unsuccessful in securing a good result and finally a fusion operation was performed. In the remaining case, the open re-

duction was accompanied by a plastic operation on the head of the femur and new bone formation developed, which accounted for the poor result. Of the 3 hips which became ankylosed, 1 had the two-stage operation with release of all structures from the neck and trochanters plus heavy traction preliminary to the reduction and the third also had a plastic operation on the head of the femur, and it was stated in the operative note that very poor cartilage remained.

Shelf Operations Alone

Of the hips which were treated by a "shelf operation alone," of which there were 16, no cases were considered as "excellent", but 8 hips or 50 per cent were recorded as "good" end-results; 4 hips or 25 per cent were considered "fair"; and 4 hips or 25 per cent had "poor" end-results. An analysis of the "poor" end-results showed 1 hip with a considerable amount of new bone formation about the upper margins of the femoral shaft and the patient complained of pain and disability which was more marked after operation than before; another hip, in a patient 40 years of age, was treated by a plastic operation on the femoral head as well as a shelf, and the result was marked limitation of motion plus pain, so that eventually an arthroplasty was performed; in the 2 remaining cases, the dislocations were due to infantile paralysis and in both instances the formation of a shelf failed to stabilize the hip joints, and in 1 of these cases an arthrodesis had to be performed.

Summarizing these statistics in a little different way, it might be stated that good functional results were obtained in 88 per cent of the closed reductions; in 27 per cent of the open reductions; in 25 per cent of the "open reductions plus shelves"; and in 50 per cent of the cases having shelf stabilizations alone.

These end-results are far from satisfactory, but it must be recognized that two-thirds of the patients in this study were over 5 years of age when treated and that perfect results are impossible to obtain in individuals of more advanced age.

CONCLUSIONS

1. Closed reduction in infants gives promise of the best end-results in congenital dislocation of the hip, and early diagnosis is the most important step toward this end.
2. In order to obtain better results in the treatment of congenital dislocation of the hip we must eliminate entirely those procedures that entail the use of force or undue trauma.
3. In every instance where undue force was employed in securing reduction, the end-result was disappointing and function sacrificed.
4. Traction by the Hoke method or with a Kirschner wire, preliminary to reduction, was effective in drawing the femoral head down opposite the acetabulum in practically all of the cases in which it was tried.
5. Open reductions that required instrumental leverage or heavy traction usually resulted in marked limitation of motion or ankylosis.
6. Stripping of the neck and trochanters of the femur, to gain length and secure reduction, ended disastrously in every case, so far as future motion in the joint was concerned.
7. Plastic operations on the femoral head or on the acetabulum should never be done without the knowledge that function will be severely impaired thereby rather than improved.
8. Shelf operations have proved their worth in the treatment of congenital dislocations of the hip and it would seem a wise precaution to add this procedure in all cases of open reduction.
9. Nothing conclusive can be stated from this small series of cases as to the relative merits of the different types of shelf operations.
10. Shelf operations alone are rarely successful in infantile paralysis cases where the hip muscles are involved.
11. Were we to formulate any general rules from our experience as to the optimum time and method of operation in these cases, we must conclude that:
    1. Gentle closed reduction should be attempted in all children under 3 years of age. If reduction fails, or if the hip is not secure after six months in plaster, open reduction is indicated.
    2. In children over 3 years of age, open reduction is advised, and reduction accomplished if at all feasible. However, the decision of whether to reduce the hip or stabilize it unreduced by a shelf, should be made at the time of operation. Should reduction necessitate the use of force, or instrument leverage, or mechanical traction, it is contraindicated, and a shelf stabilization should be substituted as the procedure of choice.
    3. In individuals over the age of 9 to 10 years, the shelf stabilization operation alone is indicated.

END-RESULTS OF TREATMENT IN CONGENITAL DISLOCATION OF THE HIP

	Number of Cases	Excellent	Good	Fair	Poor	Ankylosis
Closed reductions	16	10	4	2	0	0
Open reductions	15	1	3	5	4	2
Open reductions plus shelf	8	0	2	1	2	3
Shelf operation only	16	0	8	4	4	0
Total	55	11	17	12	10	5

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CAFFEINE INTOXICATION

Report of a Case the Symptoms of which Amounted to a Psychosis

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CAFFEINE is a trimethylxanthine compound which for many years has been successfully and widely employed as a drug and as an article in diet. It is a purine derivative and is closely allied to purine bases (xanthin, adenine, guanidine) and to commercial adrenalin, which is in turn related to incret (epinephrine, Abel) of the suprarenal glands of vertebrates. It is also closely allied to other xanthin bodies found in the urine and tissues of animals.

The pharmacologic action of caffeine can be summarized as follows:

(1) It stimulates the central nervous system.

(2) It increases muscle irritability, absolute strength and extensibility in small doses, and in large doses produces muscle stiffness and hardness.

(3) It produces no important change in pulse rate, volume or blood pressure in ordinary doses, but in large intravenous doses the heart is accelerated to final weakness and irregularity, the blood pressure rises slightly, and the peripheral blood vessels dilate.

(4) It quickens respiration, making it more shallow but aerating the blood better.

(5) It increases the body temperature slightly.

(6) It increases the secretion of urine over and above the total intake, decreasing thereby the various body fluids.

According to Cushny[1] the stimulation of the central nervous system by caffeine is particularly evident in that part associated with the higher psychic functions. "The ideas become clearer, thought flows more easily and rapidly, and fatigue and drowsiness disappear. Not infrequently, however, connected thought is rendered more difficult, for impressions follow each other so rapidly that the attention is distracted, and it requires more and more effort to limit it to a single object. If the quantity ingested is small, however, the results are of distinct benefit in intellectual work. The capacity for physical exertion is also augmented, as has been demonstrated repeatedly by soldiers on the march and, more recently, by more exact experiments with the ergograph. The stimulation of the higher nervous centers is often manifested in the insomnia and restlessness which in many people follow indulgence in coffee or tea late at night. Kraepelin[2] has investigated the effects of caffeine from the psychologic point of view and finds that both tea and coffee facilitate the reception of sensory impressions and also the association of ideas, especially in fatigue, while the transformation of intellectual conceptions into actual movements is retarded. This he regards as due to stimulation of the highest or controlling functions of the brain, caffeine acting on the same parts as are first affected by alcohol and the methane derivatives, but altering them in the opposite direction. The effect of caffeine on the acuteness of the senses has been demonstrated by the greater accuracy of touch under its influence.

"Large quantities of caffeine often cause headaches and some confusion and in rare cases of special susceptibility a mild form of delirium may be elicited, or noises in the ears and flashes of light may indicate derangement of the special senses. The pulse is quickened, and occasionally palpitation and uneasiness in the region of the heart are complained of. Convulsive movements of the muscles of the hand and tremor in different parts of the body have also been recorded in some cases."

Thus, caffeine when used or prescribed intelligently is of definite value in human economy. When used or prescribed ignorantly

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or carelessly, it is probable that sooner or later intoxicating effects will manifest themselves.

It is the purpose of this paper to present a case in which caffeine in both small and large quantities was able to produce such an abnormal mental state as to necessitate hospitalization.

CASE HISTORY

The patient (W. M., No. 33,907), a twenty-four year old, white female, was admitted to the hospital because she was excited, noisy and had a tendency to "stereotypy in her emotions."

Family History—The paternal grandfather deserted his wife and she, a dominating, strong-minded person, brought up her family alone. The patient's father, although a large man, is constantly intimidated by his wife who has a violent temper and rules the family to the point of periodically driving them from the home. At times she is very abusive verbally and physically and makes extremely unjust accusations. The patient is the second of seven siblings. The oldest, a boy, had rheumatic fever and chorea. The third, a girl, died of a brain tumor. The remaining four are girls and all are well. A maternal uncle was a patient at a mental hospital and eventually died from a cut throat. A maternal aunt had a short period of depression at the age of forty-five years.

Personal History—Her birth and early development were normal. She had the usual childhood diseases. At the age of nine years she had epidemic influenza at which time she was quite ill. After this she had rheumatic fever from which she retains a rheumatic heart. She has had several subsequent debilitating attacks of rheumatic fever. In 1933 she had an appendectomy after which she developed menorrhagia and metrorrhagia. Her periods have occurred every two weeks with excessive flowing, whereas prior to that and since their onset at the age of fourteen years they were regular. Her personality make-up varies with the informant. Her mother describes her as an irritable, nervous, high-strung and defiant person; her father describes her as being perfectly normal. Her friends say she is friendly, sociable, cheerful and a fairly good mixer. Her employer describes her as steady, likable and dependable.

Present Illness—In the spring of 1935, because of persistent fatigue and exhaustion which had lasted for three years and which was interfering with her working efficiency, she went to a general hospital for observation and treatment. The interne to whom she was assigned told her that there was nothing wrong with her and that she was psychoneurotic. She accepted his statements and advice and began to force herself to keep active in spite of her fatigue. During the fall and winter of 1935 her feelings of fatigue and exhaustion increased. On the advice of this same individual she began taking caffeine citrate, gr. 1½, three times a day. This made her feel more energetic, but she became nervous, restless and could not sleep at night. For her insomnia she was given phenobarbital.

On February 25, 1936, she was planning to attend a party. In order to give herself a feeling of well-being and to "pep" her up so that she could enjoy the party, she took several of the one and a half grain caffeine citrate tablets. Shortly afterwards she became silly, elated and euphoric. As hours passed she consumed more and more of the tablets until before the party started she had taken the contents of the box—forty tablets, sixty grains. She became confused, disoriented, excited, restless and violent, shouted and screamed and began to throw things about her room. She became exceedingly profane. Finally she collapsed and was removed to a general hospital where, on admission, she spoke in a "somewhat manic way and was very uncommunicative". She was "in some distress, talking rapidly with flight of ideas." Her physical examination showed a well-developed and well-nourished girl with a few purpuric areas of skin. Her heart was enlarged to the left; the rate was rapid and the rhythm regular, with a rough systolic murmur at the apex. The liver edge was palpable; there was tenderness in her midsternal line. The knee jerks were diminished. The blood pressure was 110/65, the temperature 99°, the pulse 95 and the respirations 20. Laboratory examinations: 8 urines were negative except for a very slight trace to a slightly perceptible trace of albumin in four and 10-15 to 3-5 white blood corpuscles in three, specific gravity to 1.025; the blood contained 4,800,000 red blood corpuscles with a hemoglobin of 90 per cent. There were 7,800 white blood corpuscles, but on February 26, two days later, they started to rise, reaching 13,300 on March 10, with a fall to normal one week later; the platelet count was normal. On February 26 it was stated that, "Patient has had a difficult situation financially and in her relationships with the nuns, boys and so forth. Impression: Hysteria." During the patient's stay in the hospital, from February 25 to March 26, her psychosis gradually subsided. Those caring for her felt that the "patient's behavior was due to a conflict between her intense religious fanaticism and her occupation as a check-room girl with its associations and temptations." After her first week the manic state had completely disappeared, but she was still very unco-operative and very unmanageable. During the second week numerous ecchymotic areas appeared on various parts of her body. Bleeding and clotting times were 3½ minutes and 3 minutes, respectively. The tourniquet test was negative. Sedimentation rate was low and the white count was slightly elevated. These ecchymotic areas it was believed were "probably due to an abnormal capillary fragility which was normal for the patient." During the middle of the third week she began to run an elevated temperature up to 99.5° which fell again to normal toward the end of the fourth week. No explanation for this temperature was found. She was then discharged with the psychiatric diagnosis, psychoneurosis, anxiety type, with a hysterical episode.

Her next episode occurred on April 1, shortly after she had recovered from an attack of rheumatic fever. Again because of her feeling of fatigue and exhaustion she took caffeine citrate to make her feel better. After taking a few of the one and a half grain tablets she rapidly became confused, disoriented and irresponsible and before long had taken the entire contents of the box—fifty tablets, seventy-five grains. She became unmanageable, screamed, laughed and cried. She was admitted to a general hospital in "an irrational state varying from wild, manic screaming, kicking and biting, to muttering semi-stupor." Physically she showed a few purpuric spots on her skin, her heart was enlarged to the left, rapid and regular, with a rough systolic murmur at the apex. The liver edge was palpable and there was exquisite tenderness at the midsternal line. Her blood pressure was 110/65; temperature 97°; pulse 86; and respirations 28. Blood examination showed 4,680,000 red blood cells and 12,400 white blood cells with a hemoglobin of 89 per cent. The differential count was normal. A consultation stated, "Hysteria without question." From April 1 to April 3 she "failed to show symptomatic improvement while on the ward and continued wildly manic." It was felt that her condition "might be hysteria rather than a serious psychosis." She was given the psychiatric diagnosis, acute manic

hysteria, questioned early manic-depressive psychosis and psychoneurosis, anxiety type, with a hysterical episode. On April 3 she was sent to the Psychiatric Clinic of the Boston State Hospital.

On admission she appeared as though she were intoxicated. Her speech was thick and somewhat incoherent. She could not be induced to stand or walk. In bed she tossed and rolled so that she was in danger of falling out. She was resistive and it was necessary to restrain her.

Physical examination showed her to be a tall, pale, asthenic and well-nourished girl with several areas of ecchymosis on her body. The pupils of her eyes were small, round, equal and regular and reacted to light. The fundi were normal. She complained of difficulty in seeing and of cloudy vision. Her ears, nose and throat were negative. Her mouth and tongue were dry. Her lungs were normal. Her heart was slightly enlarged to the left. There was a soft blowing systolic murmur heard over the mitral area and transmitted to the left. The blood pressure was 110/70. There was no evidence of sclerosis of the vessels. The abdomen and genitalia were negative, although she complained of backache and pain on voiding. The extremities were normal. Neurologic examination showed all her reflexes, both deep and superficial, to be hyperactive. There was no muscle twitching. She was extremely hyperesthetic; the slightest touch caused her very definite pain. The temperature was 99°; pulse 70; respirations 20.

Laboratory examination this time showed: Urine—straw-colored; specific gravity 1.026; acid; slight trace of albumin; ammonium urates and a few epithelial cells in the sediment. Blood—hemoglobin 80 per cent; red blood cells 4,300,000; white blood cells 10,300; differential neutrophiles 62 per cent, large lymphocytes 22 per cent, small lymphocytes 12 per cent, monocytes 2 per cent and eosinophiles 2 per cent, with platelets abundant, slight achromia, poikilocytosis and anisocytosis. Clotting time 5 minutes. Hinton reaction negative on blood serum. Basal metabolism rate, —4 per cent.

Mental examination showed a young girl lying in bed in restraint. She appeared to be of average intelligence and an intelligence test given about four weeks later confirmed this (I. Q. 93, M. A. 14 years, 10 months—normal intelligence). She still appeared narcotized, her speech was thick and her eyes rolled up. Her flow of thought and speech was rambling and she often lost the thread of the conversation. She was confused and answered questions incompletely, showing some distractibility, but no true flight of ideas. Her psychomotor activity was increased in a purposeless manner. She pulled at the restraint and tossed about continuously. She cried out and screamed. Her emotional tone was one of depression and anxiety. There was no euphoria or sense of well-being. Her mental content could not be obtained, but she did not appear to be hallucinated, nor did she react to any delusions. She was correctly oriented for person. She knew the year but not the day of the month. She thought she was in prison. Her memory could not be adequately tested because of lack of co-operation, but she gave her own history in a fragmentary way but not chronologically. Her attention wandered so that general information could not be obtained. She was so confused that she could not do simple calculations. She had partial insight into the cause of her condition and the oddity of her behavior, but her judgment was impaired.

Clinical Course—During the first day or two of her stay it was necessary to restrain her because of her tossing and rolling. In spite of this she was extremely drowsy and her speech was very thick. She slept most of the time, but could easily be roused. On each succeeding day, however, there was a very definite and rapid improvement in her condition so that on April 10 she was mentally clear and could give an excellent account of herself. Her vision was entirely normal. She was definitely emotionally unstable, crying, laughing and giggling easily. She felt weak and exhausted and had to remain in bed. By April 17 she was sitting up in a chair and was stable emotionally except for a mild depression. She had excellent insight into her condition. On April 25 her B. M. R. was —12 per cent. By May 3 she had entirely recovered from her psychosis and was feeling quite well. However, she was felt to be somewhat emotionally unstable in that she was easily moved to pity, sympathy, devotion or cynicism and her feelings were easily hurt. On May 22 her hemoglobin was 80 per cent and her red blood cell count 4,400,000. Her basal metabolism rate was +10 per cent. On May 26 her hemoglobin was 74 per cent, her red blood cell count 4,360,000 and her white blood cell count 9,050, with neutrophiles 68 per cent, small lymphocytes 21 per cent, eosinophiles 1 per cent and transitionals 10 per cent. Shortly after this she was observed to be rather silly, laughing and giggling. She was somewhat nervous. Investigation showed that she was drinking coffee, four cups a day, to overcome her feelings of fatigue—this in spite of her insight and of therapeutic interviews in this respect. Coffee and tea were removed from her vicinity and soon she again became entirely normal and was dismissed from the hospital.

DISCUSSION

Caffeine intoxication is a state of poisoning which is not often encountered; this is particularly true when the vast amount of caffeine which is used for medical purposes and in articles of diet and refreshment is considered. The literature on this subject, moreover, is not very abundant. Much has been written in a general and from a purely neurologic[3] or didactic psychologic[4] point of view — all or most all of the work being controlled or partially controlled laboratory work. The psychiatric aspects of caffeine intoxication are extremely meager and have been neglected.

In 1914, Orendorff,[5] in a letter to the Editor of the *Journal of the American Medical Association*, described the case of an eighteen year old girl who was slightly frail of stature and of frail physique. For four years she had had ordinary symptoms of asthenopia, which had been worse for several months. Correct eyeglasses gave no relief. She "became eccentric, stubborn, at times exhilarated, then depressed, and would exhibit lapses of memory and deportment with indifference to the usual conventionalities and proprieties." Headaches were intermittent. She had insomnia at night and would fall asleep during the day at her work. Investigation of her case showed that she was drinking three to six glasses of coca-cola a day and two to three cups of strong coffee at mealtimes. Curtailment of the amount of daily caffeine ingestion caused prompt improvement in all symptoms.

In 1920 Brandenburg[6] described and sum-

marized the symptoms of the coffee addicts he had observed as vertigo, anxious feelings, weakness, weak spells, agitation and a heavy feeling of the heart.

In 1925 Powers[7] reported the symptoms of twenty cases of coffee intoxication, stating that he had observed thirty-six. The symptoms occurred as follows: vertigo twelve cases, headache eleven, nervousness eleven, visual disturbance six, insomnia four, nausea three, tinnitus three, cardiac arrhythmia two, abasia two, projectile vomiting one, acrosyncope one, and anginoid pains one. He describes the syndrome of coffee intoxication as vertigo, headache, scotomata, and nervous irritability, with sometimes other symptoms produced by toxic effect on various parts of the nervous system, either peripheral or central. Powers reported two interesting cases. In one there was a rhythmic tremor of the left eyelid, twitching of the facial muscles on the left side and tremor of the tongue and hands. When coffee was removed from the patient's diet the symptoms disappeared. In the second case there was headache, cloudy vision, nervousness, weakness and feelings of constriction of the throat for about a year. She was treated as a psychoneurotic without any benefit, but when she refrained from drinking coffee her difficulties disappeared.

The case which we have presented is a fairly clear cut one of caffeine intoxication, the syndrome having been repeated several times and the relationship between the caffeine and the psychosis established. The symptoms of the psychosis were essentially those of the manic-depressive group with interspersed elements of a delirium and confusion. Some neurotic elements were likewise present but were not unusual. Her fatigue and exhaustibility were certainly not psychoneurotic and her excitement was certainly not explainable upon a hysterical basis. This being true, some sensible speculation of the mechanisms involved is reasonably justified.

The family history of the patient both remote and immediate is definitely pathologic. Her parents were sufficiently badly matched that a most unpleasant home situation existed and against which the patient, endowed with an unstable constitutional make-up, reacted in a very sensible manner. Instead of trying to fight the situation or to get along in it, she left home and attempted to earn her own living. She did this rather well even though somewhat handicapped by her rheumatic heart which incapacitated her at intervals and which induced in her a lessened capacity for work. As a result of this lessened capacity and a position which entailed much lifting and standing, she was fatigued and exhausted at times—sometimes so much so that she became nauseated and vomited. This fatigue and exhaustion were wrongly diagnosed as symptoms of a psychoneurosis, which resulted in poorly prescribed therapy, such as for her to "push herself" and to take caffeine to "pep her up". When the caffeine had produced nervousness and insomnia, phenobarbital was prescribed to permit her to sleep. This continued until she was so intoxicated from caffeine, and possibly from the barbiturate, that she became confused and, in her confusion, took enough caffeine to precipitate a psychosis severe enough to necessitate hospitalization upon two different occasions. The second psychotic episode, in the opinion of the writers, was gross evidence of poor judgment in a girl of normal intelligence and good insight.

The real problem at hand is, why did the girl react so easily to caffeine? There is some additional information which helps to clear this up. In interviews with her it was found that at one time bromides were prescribed after she had had an attack of sore throat and rheumatic aches and pains. Whenever she took the prescription she would become cyanotic and depressed and stagger as though she were drunk. Aspirin which she occasionally took for headaches would make her feel silly and light-headed. Smoking cigarettes would lessen her fatigue somewhat. An ordinary alcoholic drink would intoxicate her. A cup of coffee or tea would diminish her fatigue, make her feel better and stimulate her. These facts alone are sufficient to make one feel that this girl is abnormally sensitive to drugs of many sorts, and the type of sensitivity would indicate a very unstable autonomic nervous system.

Further investigation at the Clinic showed that she was not only sensitive to drugs but also markedly sensitive to certain proteins. She gave a strongly positive reaction to the von Pirquet test, so much so that a chest plate was immediately taken. Her Schick test, as well as the control, was strongly positive. Such additional marked sensitivity caused us to search for other evidence which might indicate that the entire basis might be allergic.

At the 1936 meeting of the Association for Study of Allergy, Squier and Madison[8] pointed out that allergy is apparently one cause of thrombocytopenic purpura and that some persons get the disease from certain drugs to which they are peculiarly sensitive. Vaughan and Pipes[8] reported that certain allergic patients have stomach upsets following contact with the allergic substance and Gay[8] pointed out that allergy may be the cause of unexplained fever which persists in some individuals for no apparent cause. The symptoms mentioned by these workers and other closely related ones that she had suggest that she was definitely allergic. Such symptoms were the purpura which ap-

peared suddenly, the menorrhagia and metrorrhagia which probably followed intra-abdominal bruising, increased capillary fragility, episodes of unexplainable temperature rises, unexplainable vomiting, headaches, unexplainable episodes of leucocytosis and liver enlargement and tenderness.

When all of the findings are assembled it certainly appears that we have: (1) A young girl, poorly endowed constitutionally (unstable), with emotional lability, who is made more so by the debilitation following rheumatic fever. (2) Marked evidence of drug sensitivity and protein sensitivity. (3) Physical and laboratory evidence of allergy.

The problem which brought the patient to the hospital was an active psychotic state. This state, it so happened, was the reaction of the total personality of the individual to caffeine which constituted a part of her total environment. This part environment constituted a substance to which she responded in an allergic manner, and this response manifested itself more strongly as a psychosis than in any other manner. When the incompatible environment, caffeine, was removed, she immediately returned to her former normal state. Such an allergic basis for a psychosis is a bit unusual, but in this case the facts certainly warrant such conclusions.

It was extremely unfortunate that this girl was diagnosed a psychoneurotic, for it was unquestionably this diagnosis which led to the treatment which in turn produced the actual psychosis. Such an instance is probably not an isolated one. The diagnoses, psychoneurosis, neurosis, hysteria and such, are labels which come freely and easily to the minds of medical men when they have a diagnostic problem which baffles them. This group of diagnoses might well be labeled the "waste basket of medicine," for it is certainly true that the future physician is, in all but a very few medical schools, inadequately or not at all instructed in the field of psychoneuroses, that the practicing physician, as a rule, not knowing the intricate workings of the psychoneurotic mind, does not attempt to remedy that deficiency and that when ordinary diagnostic measures fail, an acute emergency not arising, the physician, being pushed for time, is very prone to take the easiest way out—a diagnosis must be made, so the patient becomes psychoneurotic. Thus, it is highly probable that in the great population already labeled psychoneurotic there are many, who, if investigated carefully from a medical point of view, would be able to be correctly diagnosed. Such correct diagnosis having been made, it is possible that, as in our case, some simple form of treatment would relieve what had apparently been a malignant psychoneurosis.

SUMMARY

A case of caffeine intoxication has been presented. It has been pointed out that this intoxication with its resulting complications was probably explainable upon an allergic basis.

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REVERSION OF CARDIAC ENLARGEMENT IN A FOUR YEAR OLD CHILD FOLLOWING TREATMENT FOR AVITAMINOSIS

BY LOUIS RABINOWITZ, M.D.,* AND EDWARD J. ROGERS, M.D.*

IN children with organic heart disease, cardiac enlargement is almost always permanent and irreversible. But there are several conditions where heart enlargement may recede following proper treatment.

Walker, in a recent paper, mentions arteriovenous aneurysm, beriberi, and myxedema as three conditions wherein the ensuing cardiac enlargement may disappear after treatment of the respective disease process. To these, a correspondent added that the heart enlargement secondary to pulmonary tuberculosis receded following intensive diuretic therapy (Rosenblatt).

The case presented is that of a four year old male child with chronic avifaminosis, and cardiac enlargement which disappeared following adequate treatment of the avitaminosis.

CASE REPORT

R. H., a four year old white male child, entered the Caverly Preventorium for Children October 19, 1935, with a chief complaint, as stated by the informant, his mother, of anemia, prostration, and fever for several months.

The patient was born in Vermont, September 14, 1931, at the eighth month of gestation and weighed nine pounds. At the time of his birth his mother was 33 years old and his father 36 years old, this boy being their fifth child. The mother nursed the boy for the first two weeks when he was placed on cow's milk and "white Karo". While the baby was not sick, he did not appear strong.

At about ten months of age he suddenly began screaming when his limbs were handled. The very approach of his parents to his bedside elicited outcries. The family physician examined the child at this time and "could find nothing the matter". About three weeks later the child again began to scream whenever he was touched. This time the family called a chiropractor who gave the child "adjustments". These "worked". The "large black circles" under the child's eyes disappeared. No more "spells" occurred until the winter of 1935, when the screaming again appeared. The chiropractor again "adjusted" the child and improvement was noted.

The child was late in sitting and standing. He did not begin to walk until May, 1935, about three years and nine months after birth. He never ate well, and it was not until the summer of 1935 that he had any solid food which consisted of some bread, jelly and cake—the only other food or medication being milk and "Karo syrup". The child had had measles, chickenpox, and whooping cough.

Physical examination at the Preventorium disclosed a four year old white male child, thirty inches long and weighing twenty-seven pounds. He was mildly dyspneic and hoarse. His head was abnormally shaped with enlarged temporal veins. The facies was peculiar, somewhat senile. The skin was dry, scaly, and of a markedly lemon tint. Petechial spots were noted at the elbow bends, and ecchymotic areas along the lower legs. Small orbital hemorrhages were present. The breath was foul and there was evidence of delayed dentition as well as irregular enamel formation. The gums were deep bluish-purple in color and spongy about the upper central incisors. They bled easily with some tissue slough. The tongue was coated and the tonsils hypertrophied.

The pulse was rapid, small, and irregular with the presence of a transitory gallop rhythm. A precordial thrill was present. The area of cardiac dulness was markedly extended to the left axilla. Loud blowing systolic and diastolic murmurs were heard over the mitral area transmitted to the axilla and back, and entirely obliterating the heart sounds. A blowing diastolic murmur was noted in the pulmonic area transmitted down the sternum. The aortic first sound was louder than the aortic second sound, while the first pulmonic sound was softer than the second pulmonic sound.

There was general enlargement of the abdomen, and the spleen could be palpated.

Pain and tenderness were noted in all joints, and increased on motion. There were bilateral redness and swelling of the ankles with edema of the feet. The muscles were small and flabby. There seemed to be some swelling and tenderness about the lower ends of the diaphysis of the tibia and femur of both legs. When an attempt was made to stand the boy on his feet, pseudoparalysis seemed to be present. There was epiphyseal enlargement of all the long bones with a tibial tendency to curvature.

Clinical observation later disclosed a temperature rise to 102 to 104 degrees F. rectal in the afternoon. General weakness and constipation were marked. The child was very restless at night, and sweating was very profuse and general, being most marked at night and of a sour odor.

Examination of the blood disclosed a hemoglobin of 25 per cent, red blood cells of 1,950,000, and a white blood count of 5,500 differentiated as eosinophiles 0.5 per cent, basophiles 1.5 per cent, stab forms 3 per cent, segmented forms 46.5 per cent, lymphocytes 45 per cent, and monocytes 3.5 per cent. The coagulation time was increased. The red cells showed extreme achromia, anisocytosis, and poikilocytosis. The urine was essentially negative except for a high concentration of urates.

X-ray examination of the chest showed a markedly enlarged heart shadow (figure 1). The long bones disclosed a diminution of the transverse band of the distal end of the metaphyses with decidedly broadened and irregular, frayed surfaces. There were fluffy zones of calcification at the junction of the epiphyses and the shafts with smooth ground-glass appearance of the shaft proper.

The child was placed on bed rest, and a varied diet was offered him, high in vitamin C, accompanied by liver concentrate, iron, and viosterol.

Following this treatment, the temperature gradually dropped to normal, the abnormal heart sounds and the cardiac dulness receded, the skin color lost the green-yellow tint, joint tenderness, swelling, and pain disappeared, and the child began to walk.

About three weeks after entry the hemoglobin rose to 55 per cent, and the r. b. c. to 3,250,000. In February, 1936, five months later, the hemoglobin was 90 per cent and the r. b. c. 4,900,000.

The x-ray examination of the chest at this time

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showed the recession of the cardiac shadow to within normal limits (figure 2).

At the time of discharge, May, 1936, the child was in good health with no cardiac symptoms or signs.

DISCUSSION

This child had been inadequately fed all his life. He had, besides the signs of heart disease, the symptoms and signs of scurvy, rickets, and possibly beriberi.

Following four months of proper feeding with adequate amounts of vitamins B, C, and D, all the symptoms and signs of his avitaminosis disappeared as well as the cardiac signs, and re-

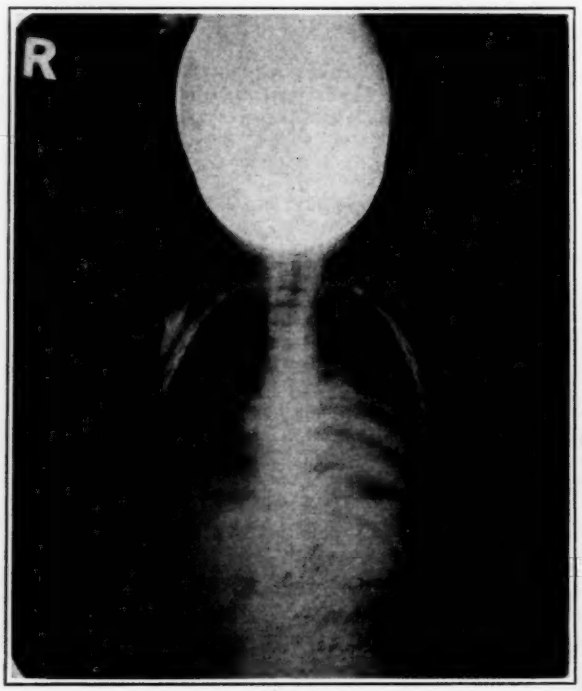

FIGURE 1. Roentgenogram of chest (6 feet) of four year old male child on inspiration. Cardiac shadow enlarged.

version to within average limits of the heart size took place.

There can be, consequently, very little doubt that the cardiac enlargement, in this case, was caused by the avitaminosis; and that treatment of the latter condition resulted in the disappearance of the cardiac enlargement noted.

The close relationship between cardiac involvement and nutrition has been fully discussed by Wenckebach. Nutritional disease can result in heart disease with its concomitant feeble circulation, palpitation, edematous extremities, and heart failure resulting in death. The left ventricle may enlarge first, followed later by the right ventricle. Temperature may be noted, accompanied by cyanosis and dyspnea.

Vitamin B lack in the form of beriberi may result in cardiac hypertrophy as a secondary manifestation of celiac disease (Lehndorff and Mautner). Even the mildest cases of beriberi may show perceptible enlargement in the roentgenogram (Aalsmeer and Wenckebach). Kugel and Stoloff noted myocardial degeneration and cardiac hypertrophy in this type of vitamin lack.

Erdheim noted that the right heart was hypertrophied in cases of scurvy in children, especially in the older children. He suggested that the subperiosteal changes in the bony cage of the thorax might inhibit respiration, and thus lead to a right heart hypertrophy and dilatation. In cases of vitamin C lack, right ventricle enlargement has been noted by Hess. The same author later suggested that polycythemia might be present in this deficiency disease. In the majority of his cases Aron noted a reduction of hemoglobin and red blood cells. Abt has very recently written that the anemia of infantile scurvy is usually chlorotic.

Pfaundler noted cardiac muscle myopathy in cases of rickets. This vitamin D deficiency may show a hypertrophy of the right side of the heart

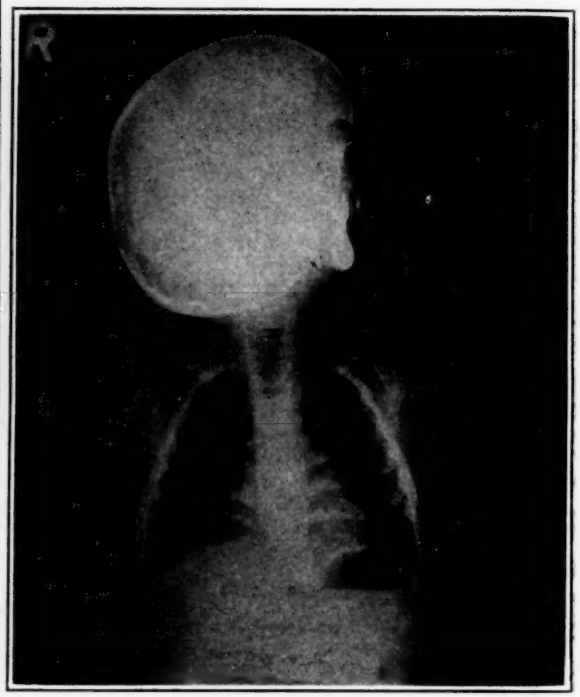

FIGURE 2. Roentgenogram of chest (6 feet) of same child 3½ months later. Cardiac shadow within normal limits.

which may last for two years and then return to normal size (Finkelstein). The cardiac enlargement in the case presented here began to recede within a few weeks after treatment had been started. Meixner noted that the left ventricle was enlarged and that the endocardium was thickened in cases of rickets.

Very interesting and suggestive experimental work has been produced by Rinehart, Connor, and Mettier, and verified by Stimson, Hedley,

and Rose, who induced rheumatic endocarditis in scorbutic guinea pigs by injecting them with a streptococcic strain of organisms, with a resulting pathology very similar to the rheumatic endocarditis in man.

SUMMARY

(1) There is presented a case of chronic avitaminosis in a four year old child exhibiting cardiac enlargement.

(2) The cardiac enlargement, and symptoms and signs of heart disease, disappeared after four months of adequate feeding.

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A STUDY OF FOUR HUNDRED JUVENILE DELINQUENTS
(Statistical Report)

BY WINTHROP B. OSGOOD, M.D.,* AND CARL E. TRAPP, M.D.*

THE Boston State Hospital has one of seventeen clinics in Massachusetts engaged in the examination of juvenile delinquents prior to commitment. Massachusetts is the second state to establish this unique juvenile law, preceded only by Virginia.

The following report represents four years' work in the study of juvenile delinquents, chiefly from the psychiatric standpoint. The juveniles examined are from the district courts of Boston. They comprise a selected group the members of which have, for various reasons, been considered for commitment and are, therefore, subject to the examinations listed below.

This study is under the supervision of the Department of Mental Diseases of Massachusetts in accordance with the provisions of Chapter 215, Acts of 1931. The purpose of the law is to give the presiding justice of the juvenile session information regarding the physical, mental and social assets and liabilities of the child adjudged delinquent for his guidance in disposition and, in the event of commitment, for the information of the superintendent of the institution, to which commitment is made. Notification is received from the probation officer of each juvenile court or session. Reports are made on a prescribed form and are sent to the court in each case.

The examination comprises chiefly a complete history, including the previous delinquent record. The school record is examined and an intelligence test done (Binet-Simon, Stanford Revision).† A physical and a psychiatric examination follow. The present study involves 400 juvenile delinquents examined during the period, 1932 to 1936. Of this number 328 were boys and 72 were girls. Their ages varied from eight to eighteen years.

STATISTICAL REPORT OF STUDY

Table 1 reveals the number of delinquents by age and sex. This table has also been graphed (chart 1). It is apparent that in both sexes the greatest number of delinquents who are brought before the court appear between the ages of fourteen and sixteen years. In the younger age groups the number of girls is much less than that of the boys; this may be due to the fact that, since the chief offenses of the girls are of a sexual nature, they tend to occur after the onset of puberty. This is in agreement with the findings of Healy and Bronner[1] who, from a study of 117 female offenders, determined that 94 per cent were sex offenders. A study of our table 4 will reveal that the great-

TABLE 1

AGE AND SEX OF DELINQUENTS

Age	8	9	10	11	12	13	14	15	16	17	18 Years
Number Boys	2	4	8	17	27	28	55	70	102	12	1
Number Girls	0	1	1	0	3	8	14	22	22	3	0

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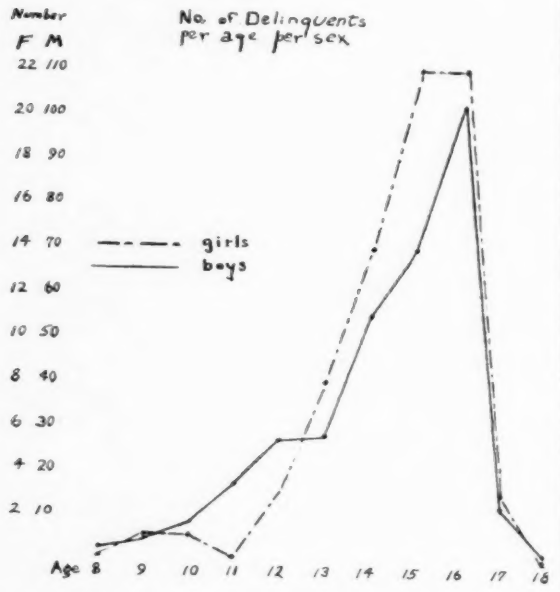
†We are indebted to Mrs. Edith James for her work in determining the intelligence ratings of these delinquents.

est number of offenses among girls were sex offenses or stubbornness.

The conception that the average juvenile

CHART 1.



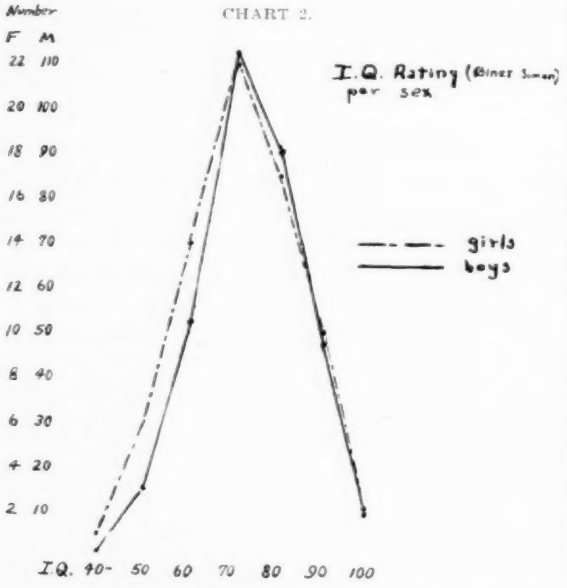
delinquent is of borderline intelligence has been confirmed in this study. (Table 2 and chart 2.)

TABLE 2

INTELLIGENCE RATINGS

I. Q.'s	40	50	60	70	80	90	100	110
Number Boys	1	15	52	112	90	47	11	
Number Girls	1	6	14	22	17	10	2	

CHART 2.



From this table can be gathered the fact that 61 per cent of the boys and 54 per cent of the girls were classified in this group. Those of normal intelligence made up 18 per cent of the boys and 17 per cent of the girls. There were 21 per cent males and 29 per cent females who were considered to be feeble-minded. The close correlation between the intelligence of the girls and that of the boys is remarkable. This is most clearly seen in chart 2.

The psychiatric examination, which is probably the most important feature, revealed that 6 boys and 2 girls were definitely psychotic. Some had been inmates previously in state hospitals or had been examined in psychiatric clinics. The mental symptoms noted in these 8 psychotic delinquents included hallucinations and delusions, abnormal emotional states and so forth. Eight boys and 6 girls were classified as of psychopathic personality. As in the cases of those who were psychotic, their offenses were serious in nature. Two boys were found to be epileptic. The results of this study are tabulated in table 3. The tabulation includes the sex, age, present offense, total number of offenses and intelligence quotient rating.

A study was then made of the total offenses, past and present, arranged by sex. These results can be found in table 4.

Among the boys, larceny was the most frequent offense. This included thievery of money and groceries and larceny of a more serious nature, such as from department stores and the like. Breaking and entering, where the delinquent had actually broken into a store or home for purposes probably of larceny, falls next in line. The unlawful use or larceny of an automobile has received a special classification and shows 147 offenses. Larceny of automobile accessories has been grouped under "auto offenses". There was only 1 charge of arson against a boy. Four boys carried weapons. Of actual assault and battery there were only 10 charges.

Among the girls it is apparent that the most frequent offenses were sex misbehavior, stubbornness and truancy. Running away from home was also frequent.

In line with this particular feature of the study it was decided to group the total number of individual offenses by age and sex. This can be seen below in table 5. At a glance it can be noted again that the greatest number of offenses occurred between the ages of fourteen and sixteen and, as might be expected, the greatest number of repetitions of offense can be noted at these ages. Thus, for example, according to the chart, at the age of sixteen among the boys, 15 had a charge of only 1 offense, 22 had already committed 2 offenses and 23 had committed 3 offenses. One delinquent had committed 8 offenses. Among the girls a similar charting can be seen. Twelve girls at the age of sixteen had only 1 offense each, 6 girls had 2 offenses and 4 girls had 3 offenses. The offenses

TABLE 3

MENTAL STATISTICS

Psychotic:

Sex	Age	Present Offense	Total No. Offenses	I. Q.	Remarks
M	13	Breaking and entering	2	79	Voices; two admissions to psychiatric clinic.
M	14	Delinquent	1	49	Hutch. teeth; depressed; speech defect; lues?
M	15	Larceny of auto	1	83	Depressed (mother's death); paranoid.
M	16	Breaking, entering and larceny	6	63	Dull; hears voices; paranoid.
M	16	Breaking, entering and larceny	1	55	Hears voices; confused; disoriented; bail $1000.
M	16	Larceny; injury to property	3	52	Has been at psychiatric clinic.
F	13	Fornication	1	79	Visual, auditory, olfactory hallucinations; paranoid.
F	15	Lewd person	1	52	Suicidal; paranoid; hallucinated.

Psychopathic Personality:

Sex	Age	Present Offense	Total No. Offenses	I. Q.	Remarks
M	14	Arson	1	93	
M	15	Larceny	1	77	
M	15	Larceny	4	85	
M	16	Larceny	1	91	
M	16	Larceny of auto	4	78	
M	16	Larceny	5	80	
M	16	Larceny of auto	6	73	Alcohol (has had D. T.'s); 10 charges of larceny.
M	18	Larceny	1	109	Also depressed.
F	13	Truant	1	82	Sex offender.
F	14	Truant	1	79	Sex offender.
F	15	Lewd person	1	69	Psychiatric clinic 1932 for examination; many sex offenses.
F	16	Stubbornness	1	99	Sex offender.
F	16	Fornication	2	96	Was at psychiatric clinic; illegitimate child.
F	16	Stubbornness	1	85	Paranoid to mother.

Epileptic:

Sex	Age	Present Offense	Total No. Offenses	I. Q.	Remarks
M	14	Runaway and larceny	1	88	
M	15	Larceny	3	80	One charge of assault; father epileptic.

TABLE 4

TOTAL OFFENSES BY SEX

Offenses	Boys	Girls
Larceny	228	6
Breaking and entering	166	2
Truancy	38	13
Breaking, entering and larceny	113	0
False alarm	11	2
Sex misbehavior—lewd person, and so forth	5	30
Assault and battery	10	0
Unlawful use or larceny of auto	147	0
Runaway from home	16	16
Stubborn child	30	41
Auto offenses	8	0
Arson	1	0
Vagrant; drunk	6	3
Carrying weapons	4	0
Forgery	1	1
Miscellaneous and unspecified delinquency	134	6

TABLE 5

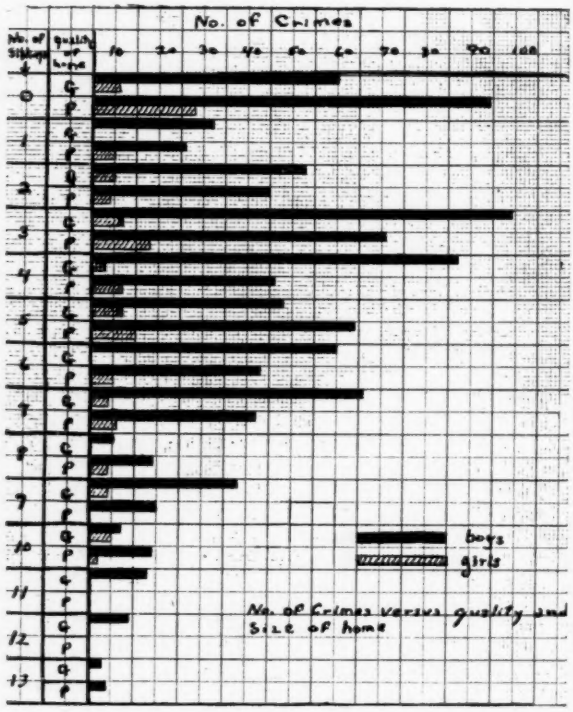
INDIVIDUAL OFFENSES ARRANGED ACCORDING TO AGE AND SEX

	Boys									Girls				
Total No. of Offenses	1	2	3	4	5	6	7	8	9	1	2	3	4	5
Age														
8	1	1												
9	1	1	1	1						1				
10	2	3	2		1					1				
11	7	4	2	2	1	2								
12	7	8	6	2	2	1				1	1	1		
13	7	7	7	6	2	1				2	3	2		
14	14	24	13	3	2		1			8	5	1		
15	10	24	18	13	4	3	1			12	6	1	1	1
16	15	22	23	19	10	3	5	1		12	6	4		
17	1	4	4	2					1	2		1		
18		1												

listed on this chart, whether the first or eighth, had all been brought to the notice of the court.

The final feature of our study was to examine the home, size of family, court records of other members of the family and so forth. This was difficult as records were scanty and much of the information came from the delinquents themselves. An examination of other members of the family in regard to court records revealed the following. Of the 328 boys, 59 had siblings with court records, 13 had fathers with court records and 3 had mothers with court records; of the 72 girls, 8 had siblings with court records, 3 had fathers with court records, and 1

CHART 3.



had a mother with a court record. As a general rule among the siblings there were minor charges of larceny and the like. Among fathers the charges varied from larceny to attempted murder. The mothers were usually arraigned as a result of sexual offenses.

A study was then made of the number of offenses as compared with the number of siblings in the family and also as to the quality of the home. This has been graphed on chart 3. Under the heading listed as no siblings, the family history was uncertain and it was difficult to determine the number of siblings, so that this actually is a miscellaneous group. Homes have been listed on the graph as good, signified by "G," and poor, signified by "P." A good home meant that both parents were living, that financial circumstances were sufficient for the care of the family, that aid was unnecessary and that there were no criminal records in the home in other members of the family. Poor homes were considered as such if the mother or father was delinquent, had a court record or in other ways was incapable of taking care of the family or if older siblings had court records and were influencing the delinquent under consideration. It is apparent that offenses in the delinquent are little influenced by the quality of the home, according to the standards mentioned above and that in many cases delinquency was greater in homes of good quality. These figures, however, can be subjected to considerable criticism, inasmuch as thorough studies of the home were difficult to make.

SUMMARY AND CONCLUSIONS

A statistical report of the study of 400 juvenile delinquents is presented, comprising four years' work. The report has included the age and sex of the delinquents, their intelligence ratings by sex, the results of psychiatric examinations, a consideration of the total number of offenses arranged by kind and by sex, a consideration of the total number of individual offenses arranged by age and sex and the condition of the home. It is apparent from this study that:

1. The average I. Q. (Binet-Simon) of the delinquents studied was of borderline intelligence rating. Twenty-one per cent of the boys of the whole group studied and 29 per cent of girls were feeble-minded.
2. Psychiatric studies revealed that 8 delinquents were definitely psychotic; 14 were classified as psychopathic personalities; and 2 were epileptic.
3. Of the offenses studied, the boys chiefly committed larceny and unlawful use or larceny of an automobile, whereas the girls had charges chiefly of sex misbehavior, and running away from home and of stubbornness. Four boys, however, carried weapons, and the most serious offense was that of arson.
4. A study of the quality of the home and the number of offenses committed shows no correlation, inasmuch as there are the same number of offenses in good and bad homes.

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CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 22401

PRESENTATION OF CASE

A twenty-two year old white man was admitted complaining of drowsiness and profuse sweating.

Two months before coming to the hospital the patient gradually developed ready fatigue, dyspnea on exertion, drowsiness, lethargy and dizziness. Previous to this time he had been perfectly well and had carried on hard physical work. At the onset of his illness he had a slight chest cough and complained on several occasions of severe cramp-like pain in the midepigastrium which had no relation to meals. Dyspnea became more marked and five weeks before entry he developed painful swelling of the ankle which lasted for about one week. At this time he went to bed and remained there until his admission. Despite the bed rest he continued to become weaker and his weight decreased from 159 to 130 pounds. He had frequent drenching sweats and felt very drowsy. Occasionally he had a shaking chill and on two occasions moderately severe nosebleeds.

For two weeks before entry he had frequent paroxysms of dyspnea while in bed, each of which lasted about ten to fifteen minutes and was associated with marked palpitation. For two days he had cough, alternating chilly and hot sensations, and a return of the cramping epigastric pain. He vomited several times. On the day of entry he became quite irritable and developed sharp pain in the right shoulder.

The patient had had acute rheumatic fever at the age of eight years, and this was followed by a "leaky heart valve".

Physical examination showed a well-developed but poorly nourished young man who appeared seriously ill. He perspired profusely and appeared to be quite restless. A small tender red area was noted in the metatarso-phalangeal pad of the right index finger. Petechiae were seen in the conjunctiva of the lower right eyelid and a few others were noted in the buccal mucous membrane. The skin and mucous membranes showed considerable pallor. The lungs were clear. The heart was slightly enlarged downward and to the left, and the apex beat was rapid and forceful. An occasional extrasystole was heard and there was a loud systolic murmur with a suggestion of a presystolic element heard at the mitral area. Beneath the sternum at the level of the third interspace a faint middiastolic whiff was heard. Gallop rhythm was noted and the second pulmonic sound was palpable, loud, and ringing in character. The blood pressure was 120/70. The liver edge extended four fingerbreadths beneath the costal margin and was soft and tender. The spleen descended to the costal margin with inspiration.

The temperature was 101°, the pulse 120. The respirations were 50.

Examination of the urine showed a specific gravity of 1.020, with a slight trace of albumin. The sediment was negative. The blood showed a red cell count of 3,140,000, with a hemoglobin of 45 per cent. The white cell count was 25,900, 80 per cent polymorphonuclears and it rose in several hours to 54,600. The vomitus gave a negative reaction to the guaiac test.

The patient suddenly died about twelve hours after entry.

DIFFERENTIAL DIAGNOSIS

DR. WILLIAM B. BREED: Unless there is some very obscure and unusual condition here I think we can at once arrive at one diagnosis. If they had had time to take a blood culture we would expect to get back a positive report of Streptococcus viridans. It is a perfectly typical story of subacute bacterial endocarditis superimposed on rheumatic heart disease. There are not very many points that we need to bring up in differential diagnosis. The sweats and fever make us think, before you go down farther in the history, of tuberculosis, lymphoma, and various other conditions. Of course, he may have one of these diseases but there is no particular point in putting them down and making a big list of differential diagnoses in this case.

There are two or three interesting points. If this man has mitral stenosis, which I think is somewhat doubtful on the evidence produced, that is an unusual condition in subacute bacterial endocarditis. Clear cut stenosis of the mitral valve is not a common finding in the presence of Streptococcus viridans infection.

Are there any comments you would like to make on the history up to the point of the physical examination which would throw any light on a reasonable diagnosis other than subacute bacterial endocarditis? Of course, we can consider perinephric abscess, subdiaphragmatic abscess, and pyelitis, but there is no particular reason for going into these conditions when there is a perfectly good history of epigastric pain, cramps, with fever, sweating, chills, nosebleeds, petechiae, and so forth.

A PHYSICIAN: Would you attempt to differentiate subacute and acute, that is hemolytic streptococcus, bacterial endocarditis?

DR. BREED: Acute hemolytic streptococcus endocarditis is a rare complication of rheumatic heart disease. I would be more willing to explain the whole picture on plain rheumatic fever rather than acute hemolytic streptococcus infection. We are always trying to decide in these cases whether there is subacute bacterial endocarditis or rheumatic heart disease with an acute flare-up of rheumatic fever. This can perfectly well be rheumatic fever alone.

A PHYSICIAN: Without joint symptoms?

DR. BREED: Yes; patients, particularly children, can die with acute rheumatic fever without having any joint symptoms at all almost as often as they die with joint involvement. To be sure, he is a young adult but I must say this is pretty rapid progress for subacute endocarditis. It usually runs longer. I am willing to introduce the simple diagnosis of rheumatic fever on this patient. Neither diagnosis can be proved on the clinical evidence. He was in the hospital only a few hours.

A PHYSICIAN: Is not fourteen years a long interval without any intervening symptoms?

DR. BREED: It is a little long but certainly not uncommon.

A PHYSICIAN: The question I meant to ask, Dr. Breed, was whether you might not elect to disregard the rheumatic side of the picture and the past history and assume that he had acute bacterial endocarditis.

DR. BREED: That is perfectly fair but I do not see how you can quite neglect rheumatic fever in the past, and known rheumatic heart disease. The superimposition of hemolytic streptococcus infection on old rheumatic heart disease is not very common, not so common as that of Streptococcus viridans, but I must admit the episode is pretty acute and short for the latter.

A PHYSICIAN: He had rather severe sepsis for a period of a couple of months, fever, chills, sweats, and so forth.

DR. BREED: It is perfectly plain to see that the consensus of opinion is beginning to point toward acute endocarditis rather than so-called subacute bacterial endocarditis.

A PHYSICIAN: How do you make a diagnosis of acute rheumatic fever without joint symptoms?

DR. BREED: In children you make it on a story of the patient's not doing well generally, not gaining weight, upon lassitude, low grade fever, slight leukocytosis, possibly a prolongation of the P-R interval by electrocardiogram, juxta-articular nodules, and by ruling out any other infection. You will see plenty of patients at the Good Samaritan Hospital with subacute rheumatic fever and rheumatic heart disease who never had any joint symptoms at all. It is the common cold that practically always precedes the rheumatic episode in children. If we can eliminate the common cold we can decrease the incidence of rheumatic fever exacerbations.

A PHYSICIAN: He was having showers of emboli. Would that speak for bacterial endocarditis rather than rheumatic fever?

DR. BREED: Yes, although one may have petechiae in rheumatic fever. He has bacterial endocarditis, I believe. I just brought up the question that it might be considered straight rheumatism because straight rheumatic infection is very severe and is often acutely fatal. But I think we all agree that he has bacterial endocarditis. The question is, what is the bacterium?

A PHYSICIAN: Is it not rather prolonged for acute?

DR. BREED: No, as a matter of fact now that we talk about it more, I am beginning to think it has too short a duration for subacute bacterial endocarditis. He may have an influenzal endocarditis. We have had a number of cases here of influenzal endocarditis with this same picture. I think we have to summarize it this way, that he probably has bacterial endocarditis and if we had him longer we would take blood cultures and perhaps make the diagnosis definite before he died. I am beginning to feel that this is more of an acute affair and that it may be due to hemolytic streptococcus or something other than viridans.

Let us go over the physical examination again. The suggestion of a presystolic mitral murmur means nothing in regard to a definite diagnosis of mitral stenosis.

A PHYSICIAN: You would expect a certain amount of stenosis?

DR. BREED: Yes, after fourteen years of the disease.

The gallop rhythm here was either due to this presystolic or late diastolic murmur, which I prefer to call it, or it indicates rather extreme myocardial damage.

The fact that the pulmonic second sound was ringing in character would lead to the suggestion that he had mitral stenosis. I think we shall find a small amount of stenosis, certainly not much. On the other hand, the diagnosis of mitral stenosis has been made on a number of children here and at the House of the Good Samaritan by different physicians on the same patient after long periods of observation and at postmortem examination the mitral valve showed no stenosis. So we are pretty chary about the diagnosis unless the signs are clear cut. However, I think we might just as well be brave and say that he has mild stenosis.

There is no indication of any aortic regurgitation here so far as the blood pressure goes, although there is a "whiff" in the third in-

terspace. It does not say whether it is right or left but it is middiastolic and therefore cannot be put into the aortic group. Therefore, I will say the aortic valve is clear as far as any organic condition goes.

The large tender liver may not be due to congestive failure; it may be due to acute infection and toxemia. If he had subacute bacterial endocarditis of the viridans type he ought to have a large spleen. This is merely just felt; and so I think we shall have to lean more toward acute streptococcus infection—on the basis of the history together with the physical examination. The question is what did he die of? It does not say under what conditions he died. He just died suddenly.

Clinical Diagnosis

Subacute bacterial endocarditis.

Dr. William B. Breed's Diagnoses

Bacterial endocarditis, ? organism.
Rheumatic heart disease with mitral stenosis.

Anatomic Diagnoses

Subacute bacterial endocarditis of the mitral valve with rupture of the chordae tendineae.
Cardiac hypertrophy, rheumatic type.
Chronic passive congestion of the liver.
Splenic infarction.

Pathologic Discussion

Dr. Benjamin Castleman: The heart weighed almost 600 grams and showed hypertrophy of both the left and right ventricles and marked dilatation of both auricles and the left ventricle. The right ventricle was about 8 mm. in thickness. All the valves were negative except the mitral, which although not very stenotic showed evidence of old infection in that the chordae tendineae were thickened. Along the edge, along the line of closure, and also extending down the chordae tendineae and up onto the auricular wall were pinkish red and gray granular thrombi, characteristic of a subacute bacterial endocarditis. The rapidity of his course may have been connected with a rupture of the chordae tendineae of the posterior leaflet due to the bacterial endocarditis.

He also had an infarct of the spleen, although the spleen itself was not very large, weighing only 370 grams. The liver was very large, over 3,000 grams, and showed congestion and well-marked central necrosis. The kidney, as is usually expected in this condition, showed an embolic nephritis. Our postmortem blood culture was unfortunately contaminated but the endocarditis was certainly entirely typical of the so-called bacterial type produced by the streptococcus viridans.

CASE 22402

Presentation of Case

A forty-seven year old poultryman was admitted complaining of ulceration of the neck, left elbow, left axilla and right antecubital fossa.

Two months before this entry the patient had entered the hospital for a period of one month, during which time these ulcerations were treated with simple boric acid and zinc peroxide dressings. They had occurred spontaneously six months previously, had remained refractory to treatment and were quite painful. Following his entry they improved very slowly but never subsided completely. No initiating trauma or causative factor could be determined. Cultures of the ulcers during this previous admission showed staphylococci, bacillus coli, and anaerobic streptococci. Biopsies were negative for malignancy and tuberculosis. Following his discharge the patient faithfully continued his treatment at home and remained quite comfortable. A week prior to readmission he changed the brand of boric acid used in his dressings, however, and shortly afterward the edges of the ulcerations became reddened and extremely tender. The ulcer on the right arm began to ache considerably and for two days there were shooting pains running up the arm.

Twenty-seven years before entry the patient had a furuncle in the left axilla which was excised but continued to drain for eight months. Fifteen years later the left forearm was amputated three inches below the elbow because of a severe hand infection following a scratch.

Physical examination showed a well-developed and nourished man with painful ulcers situated on the nape of the neck, in the left axilla, two in the left forearm stump, and one at the right elbow. All showed irregular, slightly inflamed, undermined edges with considerable induration, and raised, unhealthy looking central granulation. Small, firm, discrete, nontender cervical and axillary nodes were palpable. The heart and lungs were normal and the remainder of the examination was negative.

The temperature was 100°, the pulse 90. The respirations were 20.

Examination of the urine was negative. The blood showed a red cell count of 3,700,000, with a hemoglobin of 90 per cent. The white cell count was 9,700. A Hinton test was negative. On different occasions hemolytic streptococci were cultured both aerobically and anaerobically from the various wounds.

The patient continued to run a low grade fever ranging from 98° to 101° and although the ulcers showed some evidence of improvement the patient was considerably depressed. A determination of the patient's phagocytic index for the cultured streptococci proved to be quite

low. Attempts to obtain immune donors were finally successful and an immunotransfusion was given. This resulted in a well-defined increase of the phagocytic index but at the end of about two months the skin ulcerations again began to become painful and angry looking. Gradually his condition became worse and after about three and a half months the temperature showed a consistent daily rise to 102°. The white cell count at this time was 18,000. A pulse irregularity was shown by electrocardiogram to be resultant upon ventricular extrasystoles. No other abnormality was noted. The axillary ulcer extended beneath the pectoralis major and the pain became severe. At the end of the fourth hospital month débridement of the ulcers was performed and Dakinization instituted. Ten days later he was given a transfusion during which he had a severe chill and his temperature rose to 104° an hour later. At this time he began to vomit and complained of numbness in both legs and shortly afterward developed a flaccid paralysis with loss of all sensation in both lower extremities. Both legs were pallid and cool and there was no pulsation in the femoral, popliteal, or dorsalis pedis arteries. Suction boots were promptly applied to both legs and seven hours later the right leg was somewhat warmer and pinker than the left. Incontinence of urine and feces ensued and the vomiting continued. Two days after the acute episode the stump of the left arm became dark and cyanotic and an ulceration developed upon the lower back. The left lower limb became black and blistered up to the hip but, although pulsation, sensation, and movement continued to be lacking on the right, no other color changes became evident. The temperature rose to 103° and numerous ulcerations appeared in the mouth. The patient became progressively weaker and died six months after entry, two weeks after the onset of the acute episode.

Differential Diagnosis

Dr. Leland S. McKittrick: We can probably dispose of the first two or three paragraphs of this history very briefly. I think they form the background for what is to follow. He is obviously one of those people who have a markedly lowered resistance to pyogenic infection. I say that because of the eight months it took to heal up the furuncle, and because of his losing an arm for a bad infection of the hand. His more recent infection I assume falls into that group of cases that Meleney in New York has done so much work on, a slow progressing infection due to an anaerobic streptococcus responding to wide excision, and more recently to the use of zinc peroxide dressings, because of the oxygen that is liberated. That represents the background of this patient and only in that way does it have to do with what we are concerned with at the present time.

The next thing in his long chronic past history was his immunotransfusion with apparently some relief and then a recurrence of more rapid progression and high fever, then the pulse irregularity which the electrocardiogram showed to be due to ventricular extrasystoles. I mention that now because I do not believe it to be of much significance and doubt that we need pay much attention to it. The striking thing, that after a transfusion he had a chill, a temperature of 104°, numbness and paresis of both legs, absent pulsations from the groins down, loss or incontinence of feces and urine, vomiting, and extensive gangrene of one leg extending up to the hip, which is an extraordinarily high level for gangrene to reach on one side with absolutely no gangrene on the other side. The chill and temperature of 104° carry me back a little way to the times when we used to transfuse for pernicious anemia and transfuse some of the very late or sick pernicious anemias, and have them have a severe chill and high fever, when the blood to all intents and purposes was compatible. I do not think this is more than a severe reaction in a debilitated and sick patient. I do not believe it means an incompatible blood. I do not know just what the association is to the major episode which followed. As I interpret the early signs of numbness, absent pulsation, and cold feet, it seems that he must have had an occlusion of his aorta supposedly at the bifurcation. That is a perfectly classical picture with not so much pain as they ordinarily have, with loss of sensation, flaccid paralysis, and absent pulsations. I do not see how one can draw any other conclusion than that. The picture from this point on becomes an extraordinary one. It seems to me it is difficult to visualize how a sudden embolic occlusion of the aorta, at its bifurcation, or possibly above, could result in such high and extensive gangrene on one side and the absence of gangrene on the other side. I think one could visualize a dissecting aneurysm that exerts pressure on one side so as to occlude it completely, but permits a little blood to trickle through on the other side. There is nothing in the history to suggest this diagnosis, however, and I should be quite willing to drop it. I think it is possible that the application of suction boots may have altered the picture. It is conceivable to me that he may have been benefited on the right side from the application of the suction boots, whereas on the left side it may have done harm. We use a very tight, constricting band going around the leg in order to maintain an air-tight compartment in the boot and all of us have felt that there was some danger from the constriction of that band, and it is conceivable to me that this picture is obscured by a cer-

tain amount of benefit having been derived from the application on the one side and harm from the application on the other side. Even accepting this possibility it remains an extraordinary picture. The persistence of incontinence of feces and urine and the persistence of his paralysis on the right side lead me to believe that the occlusion extends above the bifurcation of the aorta. The fact that he had urine to pass makes me feel that unless it was a very terminal thing that the occlusion does not extend above the level of the renal arteries, because if they were occluded anuria would result. Therefore, I believe this man has occlusion of the aorta possibly extending as high as the renal arteries but not involving them. I believe that this is a little more apt to be due to a thrombus than an embolus because I do not quite visualize just where so big an embolus is coming from in a man who had no more to show in his heart than extrasystoles.

Clinical Discussion

Dr. Edward D. Churchill: Dr. McKittrick has covered the circulatory disorder admirably. I shall refer very briefly to the problem presented in the surgical care of this type of ulcer. It was a typical anaerobic or microaerophilic hemolytic streptococcus infection. The best treatment lies in dressings of zinc peroxide, a method developed quite recently by Dr. Frank Meleney of the Presbyterian Hospital. In addition, a débridement of the margins is performed to enable the zinc peroxide to come in contact with the advancing edge of the infection. The suffering of these patients is terrific. I have just looked over the daily notes and will read two or three of them. "Dressings burn a great deal." "Improvement. Patient depressed, very nervous during dressing." "Nervous, crying, a little hysterical." "Very depressed to-night. Mention made that he would be in the hospital another five or six months."

The actual débridement of the ulcer was postponed because of the very low resistance this patient showed to the organism. Our experience with immunotransfusion in treatment of infections due to this particular type of organism and lesion has not been great, nothing comparable to our experience with immunotransfusion in the pathogenic varieties of the hemolytic streptococcus, where we have had good results. Here is a note, "His wife and twelve professional donors were tested in an effort to find a donor; two days later ten more; two days later twenty-five donors studied, still unsuccessful in finding any immune." A few days later ten more were tried out of which one was accepted as an immune donor. More notes about the pains. "Perhaps some improvement but slow." "Neurosurgical consultants being solicited for possibility of relief of pain." They were not able to offer it. "Considerable pain tonight." "Patient would like irrigations spaced farther apart because of such great pain in changing." A patient in the hospital month after month with daily, painful dressings to look forward to is a terrific problem. We have seen some of these patients develop real psychoses. One patient, even after the lesion was healed, had to spend over a year in a mental institution to recover from the reaction. The mental strain of this day by day anguish wears them down like a Chinese water torture. No progress was made in combating this infection.

Clinical Diagnoses

Ulcer of the left axilla.
Embolism of left femoral artery with gangrene of the extremity.

Dr. Leland S. McKittrick's Diagnoses

Chronic ulcer (anaerobic streptococcus) of the left axilla.
Thrombosis of the abdominal aorta with gangrene of the left leg.

Anatomic Diagnoses

Chronic undermining ulcers, multiple, left axilla, back, antecubital space, and right arm.
Aortic thrombi, multiple, with occlusion at the bifurcation, and with necrosis of the wall.
Gangrene of the left lower leg.
Infarction of the spleen, multiple.
Infarction of the kidneys, multiple.
Arteriosclerosis, moderate, aortic.
Pulmonary tuberculosis, healed apical, left.
Pleuritis, chronic fibrous, apical, left.
Emaciation, marked.

Pathologic Discussion

Dr. Benjamin Castleman: The autopsy showed an emaciated man whose left forearm had been amputated. There was, of course, the huge undermined ulcerated lesion in the left axilla. In addition there were abscesses in the right antecubital space and one on the back. The cause of the gangrene of the left leg was a thrombus beginning about seven centimeters above the bifurcation of the aorta and extending into both common iliac arteries, only partially filling the right but completely occluding the left. In the proximal portion of the descending thoracic aorta about eight centimeters below the arch were two mural thrombi, one about two centimeters in diameter and another slightly smaller one. There were infarcts in the spleen and in both kidneys which undoubtedly were produced by emboli from the aortic wall thrombi.

Dr. Churchill: Can you connect it with the transfusion?

DR. CASTLEMAN: I was going to bring up the question whether the immunotransfusion may have initiated the thrombus and the second transfusion caused some of it to break off.

DR. CHURCHILL: The immunotransfusion was given in March.

DR. CASTLEMAN: I do not see how one can possibly tell whether the transfusions were responsible.

DR. MCKITTRICK: It is possible that it is the same process that we used to see in nonspecific protein therapy, where we saw thrombosis occur due to the slowing of the circulation in the prechill period, resulting in the loss of several limbs, so that we had to discontinue it or keep the limb heated up to 140°.

DR. CHURCHILL: The first transfusions he had apparently did not produce any symptoms, no chills at all.

DR. MCKITTRICK: The onset of the thrombosis occurred very comparable in time to what it did in those cases that we used to see when we were using intravenous vaccine. I saw two or three and they occurred directly following the chill.

DR. CHURCHILL: The thrombus might have been there and the transfusion could have brought on the symptoms. I do not know of any mechanism by which the transfusion would produce this. Given a mural thrombus, with partial occlusion of the vessel, a slight vasoconstriction reaction could conceivably bring into evidence the latent vascular obstruction.

We are perhaps placing entirely too much stress on this transfusion. You must realize that we had a man dying anyway of this massive ulcer.

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SERUM THERAPY OF MENINGOCOCCIC MENINGITIS

IN a recent article Hoyne[1] has recommended the intravenous treatment of meningococcic meningitis with meningococcus antitoxin and has cited the results obtained with such therapy at the Cook County Hospital and the Municipal Contagious Disease Hospital, Chicago, in support of his claims. Whereas, the fatality rate for meningococcic meningitis at the Cook County Hospital during nineteen years prior to 1934 varied from 35 to 90 per cent, with an average of 51 per cent, 201 patients since then have received antitoxin *intravenously and intrathecally* with a fatality rate of only 27 per cent. Furthermore, ninety-six patients have received only massive doses of either antitoxin or antibacterial serum *intravenously* with a fatality rate of but 16 per cent! Such results are astonishing, but require further analysis in view of the rather radical changes from the accepted method of treatment.

What evidence has been submitted to prove that the antitoxin is a more effective therapeutic agent than the usual antimeningococcic serum? In his first article on the use of antitoxin, Hoyne[2] reported results that were very favorable to its use. Although the alternate case method was not used, one case out of about four received antitoxin. The fatality rate for the cases treated with antitoxin was 24 per cent and for those treated with antimeningococcic serum was 46 per cent. It should be noted that the treatment of the former group was apparently somewhat more intensive than that of the latter, as the total dosage averaged 162 and 124 cc., respectively, and that a higher percentage of individuals with a poor prognosis—over 20 years of age—were included in the latter group.

In England, Banks[3] has reported a fatality rate of 35 per cent in twenty-six cases treated with antitoxin and a rate of 61 per cent in thirteen comparable cases treated with standard antimeningococcic serum. In Hoyne's present series which included only intravenously treated cases there is little to indicate that the antitoxin is more efficacious than serum, in spite of the fact that the average dosage of the former was 660 cc.* and that of the latter was 363 cc. Of the patients under 20 years of age, in twenty-six antitoxin treated cases there were no deaths and in seventeen serum treated cases only one: whereas, of the patients over 20 years of age, there were two fatalities among five antitoxin treated cases and only four among eighteen serum treated cases.

What evidence has been submitted to prove that intravenous therapy alone is more efficacious than combined intravenous and intrathecal therapy? Hoyne's combined mortality rate of 16 per cent is remarkably low, but the results of the combined therapy on a group of comparable cases are not given and the fact cannot be overlooked that Tillett and Brown[4] have reported a series of nineteen proved cases in Baltimore treated during 1935 by the combined method with only one death—a fatality rate of 5 per cent. Furthermore, nearly half of the patients of the latter group were over 20 years of age and the average doses of serum were 44 cc. intravenously and 117 cc. intrathecally.

Until more evidence in favor of meningococcus antitoxin and in favor of intravenous therapy has accumulated, it would seem wise for the physician to employ the standard method of treatment. Early in the disease, intravenous and intrathecal antimeningococcic serum should be administered every six or eight hours. Later in the course of the disease the route and frequency of the injections should be governed by the condition of the patient and the laboratory

*The current retail cost of this amount is approximately $165.00.

examinations. There seems to be little or no evidence that cisternal and intraventricular injections are warranted except in cases with spinal block. In such instances, a single cisternal tap followed by lavage with sterile salt solution is frequently all that is required to reestablish drainage. To be effective the serum used must be capable of agglutinating the meningococcus isolated from the case in relatively high dilution. Like all other treatment with antibacterial serums, the earlier it is begun the better the prognosis.

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THEN AND NOW

THE medical fees of one hundred years ago are discussed in the *New York Times* for August 18 in its review of a book by Dr. H. B. Shafer entitled, "The American Medical Profession, 1783 to 1850." The perusal of old fee tables is always entertaining and brings us usually to the same conclusion—that the doctor's recognition as expressed by his reward has been little changed throughout the past century. The normal income for the established physician was approximately $500 per year. This was made up of fees which were somewhat larger in proportion to the total income than those of today; for instance, a tooth extraction at the patient's home, $2, at the surgeon's home, $1; the removal of an eye, $100; tonsils, $25; a polypus, $25; a tumor, from $5 to $50. The variation in the last item depended in part, no doubt, upon the size of the tumor removed. The purchasing power of the physician's $500 was probably about the same as that of $3,000 today.

The medical professors of those days were enormously well-to-do in comparison with those of today. Many of them are believed to have received as much as $8,000 per year, a proportionate figure for which is reached by relatively few of our modern specialists, and by none of our bona fide professors.

Among the methods used to minimize pain are mentioned the use of opium, water of night shade, henbane, mesmerism and strapping above the point of operation in order to numb the nerves. Noise to divert the patient was also employed. In this connection one should not forget the old naval method of a well measured tap with a padded marlinspike—probably the most efficient pain killer of them all. Nor should one trained in modern laboratory calibrations fail to appreciate the nicety of touch which could produce the proper degree of concussion to tide a patient over a painful manipulation!

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

ROOT, HOWARD F. A.B., M.D. Harvard University Medical School 1919. Physician, New England Deaconess Hospital. Instructor in Medicine, Harvard University Medical School. Address: 81 Bay State Road, Boston, Mass. Associated with him is

SHARKEY, THOMAS P. A.B., M.D. University of Cincinnati College of Medicine 1931. Member of Staff, Miami Valley Hospital, Dayton, Ohio. Address: 60 Wyoming Street, Dayton, Ohio. Their subject is Coronary Arteriosclerosis in Diabetes Mellitus. Page 605.

VAN GORDER, GEORGE W. M.D. Harvard University Medical School 1915. F.A.C.S. Attending Orthopedic Surgeon, Massachusetts General Hospital. His subject is Congenital Dislocation of the Hip. An End-Result Study with Suggestions for Improved Treatment. Page 613. Address: 264 Beacon Street, Boston, Mass.

MCMANAMY, MARGARET C. M.D. Tufts College Medical School 1930. Senior Physician, Psychiatric Clinic, Boston State Hospital. Address: Boston State Hospital, Dorchester Center, Mass. Associated with her is

SCHUBE, PURCELL G. B.S., B.M., M.D. University of Cincinnati College of Medicine 1929. Senior Physician in charge of Psychiatric Clinic, Boston State Hospital. Address: Boston State Hospital, Dorchester Center, Mass. Their subject is Caffeine Intoxication: Report of a Case the Symptoms of Which Amounted to a Psychosis. Page 616.

RABINOWITZ, LOUIS. B.S., M.D. Tufts College Medical School 1932. Assistant Medical Director, State of Vermont Sanatorium; Caverly Preventorium and Bryant Health Camp. Address: Pittsford, Vt. Associated with him is

ROGERS, EDWARD J. B.S., M.D. Columbia University College of Physicians and Surgeons 1912. Superintendent and Medical Director, State of Vermont Sanatorium. Medical Director, Washington County Hospital; Caverly Preventorium and Bryant Health Camp. Associate Professor of Tuberculosis, University of Vermont College of Medicine. Address: Pittsford, Vt. Their subject is Reversion of Cardiac Enlargement in a Four Year Old Child Following Treatment for Avitaminosis. Page 621.

OSGOOD, WINTHROP B. M.D. Tufts College Medical School 1928. Senior Physician, Boston State Hospital. Address: Boston State Hospital, Dorchester Center, Mass. Associated with him is

TRAPP, CARL E. M.D. Boston University School of Medicine 1932. Formerly, Senior Physician, Psychiatric Clinic, Boston State Hospital. Assistant in Neurology, Boston University School of Medicine. Staff Member in Neurology and Psychiatry, Massachusetts Memorial Hospitals. Address: 232 Bay State Road, Boston, Mass. Their subject is A Study of Four Hundred Juvenile Delinquents (Statistical Report). Page 623.

The Massachusetts Medical Society

STATED MEETING OF THE COUNCIL

A STATED meeting of the Council of the Massachusetts Medical Society will be held in John Ware Hall, Boston Medical Library, 8 Fenway, Boston, on Wednesday, October 7, 1936 at 11 a. m.

Business:

1. Call to order at 11 a. m.
2. Reading record of last meeting in abstract.
3. Report of Committee of Arrangements for Annual Meeting.
4. Report of Committee on Membership and Finance.
5. Reports of committees to consider petitions for restoration to the privileges of fellowship and appointment of new committees.
6. Appointment of an auditing committee.
7. Fill any vacancies in the offices of the Society.
8. *Incidental Business.*

ALEXANDER S. BEGG, M.D.,
Secretary.

Boston, September 29, 1936.

Councilors are asked to sign one of the two attendance books before the meeting. The Cotting Luncheon will be served immediately after the meeting.

MISCELLANY

THE NEW YORK PUBLIC HEALTH LABORATORY

The new public health laboratory of the Board of Health of New York City will be dedicated with formal exercises on the afternoon of October 6. Addresses will be given by Dr. Thomas Parran, Surgeon-General of the United States Public Health Service; Professor C.-E. A. Winslow, of Yale University; Dr. William H. Park, founder and director-emeritus of the laboratory; Dr. Charles Gordon Heyd, president of the American Academy of Medicine; Dr. Anna Williamson, for many years assistant to Dr. Park; Dr. George McCoy, director of the National Institute of Public Health; Dr. Augustus Wadsworth, director of the New York State Laboratory at Albany, and Mayor F. H. La Guardia. The laboratory has been built with $700,000 from funds appropriated by the Public Works Administration, of which thirty per cent was an outright grant and seventy per cent a long-term loan. The laboratory will be known as the William Hallock Park Research Laboratory, in honor of its founder.—*Science* **84**:244 (Sept. 11) 1936.

DIABETES DEATH RATE UNCHANGED FOR 1935

A recent report of Frederick L. Hoffman is to the effect that the maximum diabetic death rate was 25 per 100,000 of the population in the United States for 1935. This is higher than elsewhere and, for example, is twice as high as in Canada.

In explanation Hoffman is quoted in *The New York Times* of September 20, as stating:

"It is shown by tabulation that high diabetes death rates coincide with excessive sugar consumption. The correlation is far from perfect but nevertheless significant that the United States should show a sugar consumption considerably above the average for the world at large."

HEALTH OFFICERS' MONTHLY STATEMENT OF VENEREAL DISEASES REPORTED IN THE NEW ENGLAND STATES

JULY, 1936

This statement is issued monthly for the information of health officers in order to furnish current data as to the prevalence of the venereal diseases. The following reports were received from State Health Officers. The figures are preliminary and subject to correction. It is hoped that this will stimulate more complete reporting of these diseases.

State	Syphilis		Gonorrhea	
	Cases Reported During Month	Monthly Case Rates per 10,000 Population	Cases Reported During Month	Monthly Case Rates per 10,000 Population
Connecticut	217	1.26	140	.82
Maine	42	.50	57	.67
Massachusetts	476	1.09	548	1.25
New Hampshire	10	.20	19	.38
Rhode Island	88	1.29	53	.78
Vermont	34	.90	53	1.41

Treasury Department—U. S. Public Health Service.

COMPARISON OF DISEASE INCIDENCE IN CONNECTICUT WITH 1935 AND SEVEN YEAR AVERAGE

MONTH ENDING SEPTEMBER 12, 1936

Diseases	1936 Week ending Aug. 22	1936 Week ending Aug. 29	1936 Week ending Sept. 5	1936 Week ending Sept. 12	Average cases reported for week corresponding to Sept. 12 for past seven years	1935 Week ending Aug. 24	1935 Week ending Aug. 31	1935 Week ending Sept. 7	1935 Week ending Sept. 14
Anthrax	—	—	—	—	—	1	—	—	—
Chickenpox	5	10	4	7	4	12	2	2	3
Diphtheria	—	1	—	4	8	2	2	3	1
Dysentery Bacillary	3	4	2	4	1	5	12	9	46
Encephalitis Epidemic	—	—	—	—	—	1	1	—	3
German Measles	6	2	4	2	1	—	—	2	—
Influenza	—	1	3	—	3	—	—	—	1
Measles	10	3	6	4	7	16	5	1	4
Meningococcus Meningitis	—	1	—	—	—	1	3	1	—
Mumps	16	19	23	22	8	4	7	5	10
Paratyphoid Fever	—	—	—	1	—	4	1	4	12
Pneumonia (Broncho)	6	6	7	6	9	7	10	7	6
Pneumonia (Lobar)	6	3	7	6	7	11	5	5	6
Poliomyelitis	1	—	1	—	15	40	39	38	38
Rocky Mountain Spotted Fever	—	—	—	—	—	—	—	—	1
Scarlet Fever	3	3	7	10	11	6	13	8	22
Streptococcus Sore Throat	2	—	1	—	1	—	1	1	1
Tetanus	—	1	1	—	—	—	—	—	1
Trachoma	—	—	—	1	—	—	—	—	—
Trichinosis	—	—	—	—	—	—	—	1	—
Tuberculosis (Pul.)	18	21	21	15	24	33	12	14	17
Tuberculosis (O. F.)	—	—	3	1	3	4	—	—	—
Typhoid Fever	1	4	2	2	5	2	3	1	6
Undulant Fever	1	1	2	—	—	3	1	—	—
Whooping Cough	53	104	33	49	37	37	27	33	70
Gonorrhea	62	46	25	34	44	20	61	21	53
Syphilis	63	38	55	34	36	26	81	22	49

Remarks: No cases of Asiatic cholera, glanders, plague or yellow fever during the past seven years.

DO YOU KNOW?

The first incubator patent was issued December 27, 1870, to J. and H. Graves of Boston, Massachusetts.

Diabetes is increasing. Early diagnosis with proper treatment following enables the patient to live a useful life. Most diabetics are fat, though of course, not every fat person gets diabetes.

More injuries in athletic sports occur to the knee than to any other place in the body, according to Dr. Marcus H. Hobart, of Northwestern University.

One source of automobile accidents to people with otherwise normal eyes is a narrowed peripheral vision, which enables the driver to see only what is directly in front of him and a little to each side of his direct gaze.

General Jan Smuts said: The disappearance of the sturdy, independent minded, freedom loving individual and his replacement by a servile standardized mass mentality is the greatest human menace of our time.

The first medical school in the United States was established at the University of Pennsylvania, Philadelphia, May 3, 1765. The first doctor to receive a diploma there was John Archer, who was graduated with nine others in 1768.

Records at Bellevue Hospital, New York City, show a marked increase in alcoholism during the past year.

Don't try to scold health into your children. Example is better than precept. They see more than we think.

Insurance companies report an increase in deaths from heart disease of more than three times between 1930 and 1935. This is twice as many victims as are due to cancer, and of the number there are three times as many men as women. Doctors believe the greatest single factor is hypertension, due to strain, rush and excitement. Heart disease is the most frequent cause of death among physicians, most of whom refuse to relax the tension involved in their responsibilities, and literally die in the service of their patients, usually not unaware of what they are doing.

Shakespeare said: By medicine life may be prolonged, yet death will seize the doctor, too.—*Excerpts from the Bulletin of the New York State Medical Society.*

PUBLIC HEALTH NURSES

If the ten largest cities in the United States were ranked as to the number of public health nurses in each per 100,000 inhabitants, New York would be next to the bottom of the list. Boston is well in the lead with 45. The others follow in this order: Detroit 32, Cleveland 30, Milwaukee 30, Pittsburgh 28, Baltimore 26, St. Louis 22, Philadelphia 19, New York 18 and Chicago 17. — *The New York Times*, September 26, 1936.

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR AUGUST, 1936

Disease	Aug., 1936	Aug., 1935	5-Yr. Average*
Anterior Poliomyelitis	7	481	218
Chickenpox	118	97	109
Diphtheria	19	22	77
Dog Bite	1,063	1,117	684
Epidemic Cerebrospinal Meningitis	6	4	4
German Measles	45	89	39
Gonorrhea	562	546	644
Lobar Pneumonia	126	123	91
Measles	229	130	152
Mumps	207	225	164
Scarlet Fever	156	174	246
Syphilis	462	444	382
Tuberculosis, Pulmonary	289	319	321
Tuberculosis, Other Forms	31	35	37
Typhoid Fever	13	15	25
Undulant Fever	11	2	1
Whooping Cough	534	293	517

*Based on figures for preceding five years.

RARE DISEASES

Anterior poliomyelitis was reported from Arlington, 1; Attleboro, 1; Boston, 1; Chelsea, 1; Everett, 1; New Bedford, 1; Wilbraham, 1; total, 7.

Diphtheria was reported from Barre, 1; Boston, 7; Lowell, 3; Lynn, 5; Medford, 2; New Bedford, 1; total, 19.

Dysentery, bacillary was reported from Worcester, 1.

Encephalitis lethargica was reported from Lowell, 1.

Epidemic cerebrospinal meningitis was reported from Andover, 1; Boston, 2; Falmouth, 1; Greenfield, 1; Worcester, 1; total, 6.

Malaria was reported from New Bedford, 1.

Pellagra was reported from Norton, 1.

Septic sore throat was reported from Abington, 1; Beverly, 1; Boston, 1; Foxboro, 2; total, 5.

Tetanus was reported from Groveland, 1.

Trachoma was reported from Boston, 2; Cambridge, 1; Malden, 1; Medford, 1; total, 5.

Trichinosis was reported from Chicopee, 2; Concord, 1; Lowell, 2; total, 5.

Typhoid fever was reported from Attleboro, 1; Everett, 1; Lowell, 4; Lynn, 1; New Bedford, 2; Newton, 2; Salem, 1; Saugus, 1; total, 13.

Undulant fever was reported from Dalton, 1; East Brookfield, 1; Hardwick, 1; Holyoke, 1; Lenox, 1; New Marlboro, 1; Pittsfield, 2; Springfield, 1; West Brookfield, 1; Worcester, 1; total, 11.

Diphtheria to date is being reported seven per cent below last year's record low figure.

Anterior poliomyelitis and typhoid fever had their lowest reported August morbidity.

Pulmonary tuberculosis to date is being reported slightly below last year's figure.

Lobar pneumonia, measles, mumps, and whooping cough were reported somewhat above the five-year average.

The reporting of epidemic cerebrospinal meningitis, German measles, and chickenpox showed nothing remarkable.

Scarlet fever was reported well below the five-year average for the month.

Undulant fever continues to be reported considerably above last year's figure.

RECENT DEATHS

MINER—Harold Edson Miner, M.D., aged 54, of Holyoke and State District Health Officer for the Connecticut Valley District, died suddenly September 15, 1936, while engaged in his official duties.

Dr. Miner was a native of Holyoke and was educated in the local schools. He received his medical degree from the College of Physicians and Surgeons in Baltimore in 1905 and interned at the Holyoke Hospital. After a few years of general practice in Holyoke he was appointed as school physician to which work he devoted his entire time for eight years. In November, 1920, he joined the staff of the Massachusetts Department of Public Health, being appointed as District Health Officer and serving in this capacity until his death.

Of few persons can it be said that they were as

universally liked and respected as was Dr. Miner. Gentle of manner, eager and enthusiastic in his work, he gave unstintingly of his energies and strength to the promotion of the public health of the Western part of the State. No problem was too small to merit his attention, no obstacle too great to be overcome. The progress of public health in his district was in very large measure a reflection of his work. In his passing the State loses a public servant of the highest type, the community a staunch worker for human betterment, and his host of acquaintances a true friend whose memory will forever serve to strengthen and to comfort.

Dr. Miner is survived by his widow, Mrs. Blanche T. Miner, and five sons, Frederick, Harold, Philip, Donald and Malcolm.

BRYANT—John Edmund Bryant, M.D., a retired physician of 114 Chestnut Street, Haverhill, Massachusetts, died at his home, September 18, 1936.

Dr. Bryant was born in Brownington, Vermont, in 1876, and graduated from the Dartmouth Medical School in 1901. He was a Fellow of the Massachusetts Medical Society.

His widow and two daughters survive him.

WILLIAMS—Frankwood Earl Williams, M.D., of 44 West 12th Street, New York City, died aboard the White Star Liner, Georgic, September 25, 1936.

Dr. Williams was born in Cardington, Ohio, in 1883, and graduated from the University of Michigan Medical School in 1912. He was formerly a director of the Massachusetts Society for Mental Hygiene and an official of the Boston Psychopathic Hospital.

NOTICES

BOSTON DISPENSARY

25 Bennet Street, Boston

Medical Conference Program

Lecture Room, Second Floor
9-10 a. m., October, 1936

Tuesday, October 6—Diseases and Injuries of Knee Joints. Dr. John D. Adams.

Wednesday, October 7—Hospital Case Presentation. Dr. S. J. Thannhauser.

Thursday, October 8—Endocrine Clinic. Dr. C. H. Lawrence.

Friday, October 9—Clinical Aspects of Paroxysmal Rapid Heart Action. Dr. Samuel A. Levine.

Saturday, October 10 — Hospital Case Presentation. Dr. S. J. Thannhauser.

Tuesday, October 13—Diagnosis and Management of Pelvic Inflammatory Disease. Dr. L. E. Phaneuf.

Wednesday, October 14—Hospital Case Presentation. Dr. S. J. Thannhauser.

Thursday, October 15 — Measles Prevention. Dr. Robert W. Buck.

Friday, October 16 — Agranulocytosis. Dr. William P. Murphy.

Saturday, October 17 — Hospital Case Presentation. Dr. S. J. Thannhauser.

Tuesday, October 20 — Treatment of Gastro-intestinal Disorders in Infants. Dr. Francis McDonald.

Wednesday, October 21 — Hospital Case Presentation. Dr. S. J. Thannhauser.

Thursday, October 22—Social Service Case Presentation. Miss E. C. Canterbury.

Friday, October 23—Recent Work in Epilepsy and Migraine. Dr. William G. Lennox.

Saturday, October 24 — Hospital Case Presentation. Dr. S. J. Thannhauser.

Tuesday, October 27—The Use of the X-Ray in the Diagnosis of Pulmonary Silicosis and Asbestosis. Dr. A. W. George.

Wednesday, October 28—Hospital Case Presentation. Dr. S. J. Thannhauser.

Thursday, October 29—Some Recent Work in Diabetes Mellitus. Dr. E. A. Grossman.

Friday, October 30—Parathyroid Disease. Dr. Fuller Albright.

Saturday, October 31 — Hospital Case Presentation. Dr. S. J. Thannhauser.

MEDICAL CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 p. m. on Thursday, October 8, in the Amphitheater of the Peter Bent Brigham Hospital, Dr. James P. O'Hare, Assistant Professor of Medicine, Harvard Medical School and Senior Associate in Medicine, Peter Bent Brigham Hospital, will give a medical clinic. To it are cordially invited practitioners and medical students.

APPOINTMENT OF DR. H. D. ADAMS

Dr. Herbert D. Adams, formerly Assistant Visiting Surgeon to the Massachusetts General Hospital, has been appointed to the permanent Surgical Staff of the Lahey Clinic.

NOTICES OF MEETINGS

NEW ENGLAND SOCIETY OF PSYCHIATRY

The fall meeting of the New England Society of Psychiatry (The New England District of the American Psychiatric Association) will be held at the Brattleboro Retreat, Brattleboro, Vermont, Thursday, October 8. For further information address Harlan L. Paine, M.D., Secretary-Treasurer, North Grafton, Mass.

NEW ENGLAND DERMATOLOGICAL SOCIETY

The next meeting of the New England Dermatological Society will be held Wednesday, October 7, at 3 p. m. at the Massachusetts General Hospital, Boston.

J. Harper Blaisdell, M.D., *Secretary.*

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, OCTOBER 5, 1936

Tuesday, October 6—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Diseases and Injuries of Knee Joints. Dr. John D. Adams.

5 p. m. The Edward K. Dunham Lectureship. Harvard Medical School Amphitheater, Building C.

Wednesday, October 7—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Hospital Case Presentation. Dr. S. J. Thannhauser.

†12 m. Clinico-Pathological Conference. Children's Hospital Amphitheater.

3 p. m. New England Dermatological Society. Massachusetts General Hospital.

Thursday, October 8—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Endocrine Clinic. Dr. C. H. Lawrence.

*3:30 p. m. Medical Clinic, Peter Bent Brigham Hospital.

5 p. m. The Edward K. Dunham Lectureship. Harvard Medical School Amphitheater, Building C.

Friday, October 9—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Clinical Aspects of Paroxysmal Rapid Heart Action. Dr. Samuel A. Levine.

Saturday, October 10—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Hospital Case Presentation. Dr. S. J. Thannhauser.

*10 a. m. - 12 m. Staff Rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

October 1—Faulkner Clinical Meeting. Faulkner Hospital, 5 p. m.

October 4-17—Medico-Military Inactive Duty Training, Mayo Foundation. See page 512, issue of September 10.

October 6—Lawrence Cancer Clinic. See page 601, issue of September 24.

October 6 and 8—The Edward K. Dunham Lectureship. Harvard Medical School Amphitheater, Building C, at 5 p. m. See page 565, issue of September 17.

October 6-31—Boston Dispensary, Medical Conference Program. See page 638.

October 7—New England Dermatological Society. See page 638.

October 8—Medical Clinic, Peter Bent Brigham Hospital. See page 638.

October 8—New England Society of Psychiatry. See page 638.

October 8—Pentucket Association of Physicians. Hotel Bartlett, 95 Main Street, Haverhill, at 8:30 p. m.

October 12-16—Twenty-First International Assembly of the Inter-State Post-Graduate Medical Association. See pages 565 and 566, issue of September 17.

October 12-18—Third International Congress on Malaria. See page 1076, issue of May 21.

October 19-23—Clinical Congress of the American College of Surgeons. See page 180, issue of January 23.

October 19-31—1936 Graduate Fortnight of the New York Academy of Medicine. See page 1221, issue of June 11.

October 20-22—Academy of Physical Medicine, Annual Meeting, Hotel Statler, Boston. See page 601, issue of September 24.

October 20-23—The American Public Health Association. See page 1226, issue of June 11.

November 16—One hundredth anniversary of the founding of the Army Medical Library, 7th Street and Independence Avenue, S. W., Washington, D. C.

December 3-5—Annual Conference of the National Society for the Prevention of Blindness, Columbus, Ohio.

March 30 - April 2, 1937—First International Conference on Fever Therapy. Postponement notice. See page 52, issue of July 2.

April 21-24, 1937—American Society for Experimental Pathology. See page 1075, issue of May 21.

DISTRICT MEDICAL SOCIETIES

FRANKLIN DISTRICT MEDICAL SOCIETY

Will meet at the Weldon in Greenfield at 11 a. m. the second Tuesdays of November, January, March and May.

CHARLES MOLINE, M.D., Secretary.
Sunderland.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

October 7—Bear Hill Golf Club, Stoneham.
November 18—Bear Hill Golf Club, Stoneham.
January 13, 1937—Bear Hill Golf Club, Stoneham.
March 16, 1937—Danvers State Hospital, Danvers.
May 11, 1937—Bear Hill Golf Club, Stoneham.

KENNETH L. MACLACHLAN, M.D., Secretary.
1 Bellevue Avenue, Melrose.

PLYMOUTH DISTRICT MEDICAL SOCIETY

October 15—11 a. m. at the Moore Hospital, Brockton.

FRED F. WEINER, M.D., Secretary.
231 Main Street, Brockton.

WORCESTER DISTRICT MEDICAL SOCIETY

October 14—Rutland State Sanatorium, Rutland, Mass. 6:15 p. m.: Dinner—complimentary by the State Hospital. 7:30 p. m.: Business session and scientific program.

November 5—At 4:30, in the rooms of the Worcester Medical Library, Inc., at 34 Elm Street, Worcester, will be held the fall Censors' meeting.

November 11—Grafton State Hospital, North Grafton, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

December 9—St. Vincent Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

January 13, 1937—Worcester City Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

February 10, 1937—Worcester State Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

March 10, 1937—The Memorial Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

April 14, 1937—Worcester Hahnemann Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

May 6, 1937—At 4:30 in the rooms of the Worcester Medical Library, Inc., at 34 Elm Street, Worcester, will be held the spring meeting of the Board of Censors.

Wednesday Afternoon and Evening, May 12, 1937—Annual Meeting. Time and place for this meeting will be announced in an early spring issue of the Journal.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.

BOOKS RECEIVED FOR REVIEW

A Text-Book of Pathology. W. G. MacCallum. Sixth Edition. 1277 pp. Philadelphia and London: W. B. Saunders Company. $10.00.

An Introduction to Psychological Medicine. R. G. Gordon, N. G. Harris, and J. R. Rees. 386 pp. London: Oxford University Press.. $4.00.

A Text-Book of Neuro-Anatomy. Albert Kuntz. Second Edition, Thoroughly Revised. 519 pp. Philadelphia: Lea & Febiger. $6.00.

Medicine and Mankind. Edited by Iago Galdston. 217 pp. New York and London: D. Appleton-Century Company. $2.00.

International Clinics. Edited by Louis Hamman. Volume III. Forty-Sixth Series, 1936. 339 pp. Philadelphia, Montreal, London: J. B. Lippincott Company.

Studies from The Rockefeller Institute for Medical Research. Reprints. Volume 99. 583 pp. New York: The Rockefeller Institute for Medical Research. 1936.

Bulletin of the Neurological Institute of New York. Edited by Charles A. Elsberg. Volume 5, August, 1936. 544 pp. Baltimore: Waverly Press, Inc.

Atlas of Congenital Cardiac Disease. Maude E. Abbott. 62 pp. New York: The American Heart Association. $5.50.

Fundamentals of Human Physiology. J. J. R. Macleod and R. J. Seymour. Fourth Edition. 424 pp. St. Louis: The C. V. Mosby Company. $2.50.

Principles of Chemistry. Joseph H. Roe. Fourth Edition. 475 pp. St. Louis: The C. V. Mosby Company. $2.75.

The Surgical Clinics of North America. Volume 16, Number 3, June, 1936. New York Number. 912 pp. Philadelphia and London: W. B. Saunders Company.

A Text-Book of Physiology for Medical Students and Physicians. William H. Howell. Thirteenth Edition. 1150 pp. Philadelphia and London: W. B. Saunders Company. $7.00.

Diseases of the Air and Food Passages of Foreign-Body Origin. Chevalier Jackson and Chevalier L. Jackson. 636 pp. Philadelphia and London: W. B. Saunders Company. $12.50.

BOOK REVIEWS

Physical Therapy for Nurses. Richard Kovács. 286 pp. Philadelphia: Lea & Febiger. $2.75.

This well-illustrated "handy" book of nearly 300 pages is conservative in attitude and written by a medically trained physical therapist of wide experience and ability. The first two chapters deal with the scope and purposes of the specialty of physical therapy as a branch of medicine and with the nature of the physical forces employed in the treatment of lesions of disease and injury.

Chapter III discusses the physiological effects of the application of "heat" and the various means whereby radiant heat may be most appropriately employed.

Heliotherapy and the various artificial substitutes for natural sunlight, especially ultraviolet light, receive intelligent consideration in Chapter IV. The author states that the general "tonic" effect of ultraviolet radiation is clinically recognized. As far as we are aware, no carefully controlled clinical experiments have proved this general "tonic" effect to be either striking or constant and we fear that such a statement will give support to what has been perhaps justly termed the ultraviolet racket of the manufacturers of ultraviolet apparatus. It is probably unwise to give the impression that during convalescence from operation in infectious diseases and in bronchial asthma "light therapy has *proved* a valuable adjunct to general medical treatment". The Council on Physical Therapy of the American Medical Association, after reviewing many clinical studies and some laboratory research, has decided to reject the apparatus of all firms which make claims in advertising to the public that ultraviolet light may be expected to have any wider therapeutic value than to prevent and cure rickets and to promote the development of sound bones and teeth.

"Electricity" with a hundred pages in Chapter V is perhaps unnecessarily technical for nurses' use. The author evidently believes there is a large place for the use of the galvanic current in the convalescent therapy of poliomyelitis. This runs counter to the experience of several well-established clinics for the aftertreatment of infantile paralysis in which the method has been largely discontinued as being difficult to apply accurately to the affected muscles alone, and of doubtful value.

In discussing diathermy, the author does well to point out the possible danger of the newer form of "short wave" diathermy as contrasted with the older form of application of high frequency current, or as it is usually called, "conventional diathermy". "The operator of the short wave apparatus must depend entirely on the subjective heat sensation of the patient and has no objective means of determining excess heat either of the skin or of the deeper parts." This is especially important since in the "short wave" form more heat tends to be generated in the deeper and usually in the less conductive tissues, bones, articular cartilage and tendons, than on the more sensitive skin surfaces. The author might well have mentioned the real danger which this method may offer to the avascular and largely insensitive articular cartilage, which possesses slight power of repair and almost no power of regeneration once it is damaged. Dr. Kovács is wise in his consideration of the administration of hyperpyrexia making it clear that "artificial fever treatment in any form is a strictly institutional procedure to be applied in carefully selected cases under the direction of skilled physicians; the assisting nurses or technicians must have had ample training and must be capable of recognizing untoward symptoms and know the way to avoid dangers."

Chapters VI, VII and VIII cover fairly adequately the subjects of hydrotherapy, massage and exercise, the chapter on exercise being written by Madge C. L. McGuinness, M.D., Chief of the Clinic Department of Physical Therapy, Vanderbilt Clinic, New York City.

There follows a chapter on "Physical Therapy in Institutional Practice" with practical suggestions as to personnel, the relation of a physical-therapy to other departments of a general hospital, its desirable location and space, with a list of the equipment of the author's clinic and a record form.

A short syllabus and an index fill the remaining pages.

The book will be found quite as helpful to physicians as to nurses, although it is written as a textbook for the latter group with proposed examination questions at the end of each chapter. How technical the specialty is becoming is suggested by

the fact that, in the list of equipment of the Polyclinic Hospital's Department of Physical Therapy, twenty-one of the twenty-five pieces of apparatus listed are electrical in nature. We might have wished for one more chapter in which due emphasis had been placed upon the important contributions which may be made to the well-being and recovery of our patients by the application of the simpler and more easily grasped methods of physical therapy. This is a message which family physicians and nurses need greatly.

Clinical Heart Disease. Samuel A. Levine. 445 pp. Philadelphia and London: W. B. Saunders Company. $5.50.

This most recent book of Boston cardiology is a collection of chapters written for the general practitioner. It does not attempt a systematic survey of the whole subject of heart disease although most of the important features are at least mentioned. Rheumatic fever, valvular and pericardial disease, angina pectoris, hypertension, and the other chief etiologic agents of heart disease are considered. As a fundamental object of the book, emphasis is placed upon conditions for which truly remedial therapy exists. This leads, perhaps, to some overstressing of hyperthyroid heart disease — a subject which the author considers to be "probably the most important aspect of all heart disease", because of the optimistic therapeutic implications. Pick's disease, for which there exists similar, but far less frequent, opportunity for surgical therapy, is less clearly described, the old impression being given that it may be found with valvular disease, a large heart, and Broadbent's sign. An important cause of bloody pericardial fluid, namely, malignant disease, is not mentioned in the chapter on diseases of the pericardium. Among the less important etiologic agents, diphtheria should have been given a place, especially in the discussion of gallop rhythm; as also some word about the aberrations of glycogen storage in heart muscle as recently shown to be a possible factor in congenital idiopathic hypertrophy.

The last eight chapters are concerned with special subjects such as paroxysmal rapid heart action, acute cardiac emergencies, factors involved in dyspnea, the significance of the systolic murmur, the cardiac patient as a surgical or obstetrical risk, prognosis, and treatment of heart disease and clinical electrocardiography.

As the book has no footnotes or bibliography and does not attempt to cover the literature it is occasionally much condensed. In places somewhat more detail would be of value in instructing the general practitioner. As an example, only a few words are given about the use of acetyl-beta-methylcholine in the treatment of paroxysmal tachycardia — a drug which cannot be used freely without the occurrence of alarming reactions at times, which the physician should be ready to anticipate by having hypodermic atropine at hand.

Dr. Levine's early interest in the relationship between cryptic hyperthyroidism and heart disease resulted in the attempt to relieve heart failure, congestive and anginal, by complete ablation of the normal thyroid gland. He summarizes the present status of this procedure as follows: "The entire problem of the removal of the normal thyroid gland for intractable heart disease must still be regarded as in the experimental stage."

The chapter on electrocardiography comprises about one-quarter of the book and should serve very well to correlate clinical syndromes with their electrocardiographic explanations.

The author has been a stimulating teacher and careful observer and writer in the field of heart disease for many years. The book is to be recommended not only to students and general practitioners but also to those who are primarily interested in cardiovascular problems and wish a concise summary of his point of view.

The Art of Treatment. William R. Houston. 744 pp. New York: The Macmillan Company. $5.00.

After reading Houston's compendium, one is tempted to indulge in superlatives. Of all the books on the art of therapeutics this is the most interesting, the most thorough, the most practical. The use of the direct and forceful first person singular adds tremendously to the value of the work. The author has covered the field of treatment in a manner that is not only authoritative but he has also produced a book as readable and thrilling as the latest best seller. No words are wasted; there is a direct attack; the entire subject, including fundamentals and the minor aspects, is most adequately discussed. The patient as a whole, not as a conglomeration of separate organs and processes, is held before the reader constantly. The impacts of modern society on the mind and body of the sufferer are never forgotten. The most comprehensive functions of the physician are constantly stressed.

This is a work that should be on the desk of every doctor who wants to give his patients the very best of treatment in its widest implications. The references to the literature of medicine and its immortals show a profound cultural background that is strongly reminiscent of Osler, and his classic. A great future for this book is confidently predicted.

Strength Out of Suffering. France Pastorelli. 223 pp. Boston and New York: Houghton Mifflin Company. $2.00.

This is the life of an invalid, but a glorious adventure Madame Pastorelli has made of it. Just at the beginning of her promising career as a pianist she was stricken with a severe heart lesion. Her beloved art had been given up and her time spent in bed. But she would not acknowledge defeat. She has produced for us obtuse well folks an inspirational work that brings the blush of shame. As a pian-

ist she would doubtless have given thousands genuine pleasure, but her "Strength" goes far deeper and leaves a more lasting impression than any other form of art could produce.

Les Petites Règles de la Chirurgie Parfaite. J. Okinczyc. 60 pp. Paris: Masson et Cie. 12 fr.

This is a splendid short book, and emphasizes the necessity of paying attention to minor details in surgery. The fact that it is concise, well written and not didactic makes this short monograph worthy of an English translation. House officers in surgery would find this book very helpful. It contains much needed advice.

The following are a few of the especially interesting sentences:

(1) All surgical acts are serious things, since minor operations may lead to major complications.

(2) There are always undeserved failures in surgery, but to be sure there are also unmerited successes.

(3) Do not be too hasty in accusing the catgut or the sponges without assuring yourself that all the minor precautions were taken at the time of operation.

(4) Manual dexterity does not license one to dispense with necessary details. We are no longer in the era of bloody and rapid surgery.

(5) The progression of proper surgical manoeuvres is only a succession of imperceptible acts destined to gain one's end without traumatizing very vulnerable tissue.

(6) I will give my confidence to a fair surgeon but a good clinician, rather than to a brilliant operator who knows no clinical medicine.

A section each is devoted to sutures, hemostasis, exposure, incisions and the teaching of surgery.

A Textbook of Histology. Joseph Krafka, Jr. 246 pp. Baltimore: The Williams & Wilkins Company. $2.50.

This brief text on histology is planned for the college student with relatively little training in science. It is a readable introduction to the subject that would be of but little value to the medical student or practitioner. It might well be of service for the training of nurses.

Medical Science Exhibits. A Century of Progress. Eben J. Carey. 204 pp. $2.00.

This is a pictorial record of the exhibits contributed to the medical section of the Hall of Science at the Century of Progress Exposition, Chicago, 1933 and 1934. These exhibits were of interest in that this was the first health exhibit in any international exposition held in America. The various photographs will be of particular importance to those who have seen the exhibits and also to those having in mind a preparation of exhibits for both scientific and lay groups.

Heart Disease and Tuberculosis. Efforts Including Methods of Diaphragmatic and Costal Respiration to Lessen Their Prevalence. S. Adolphus Knopf. 108 pp. New York: The Livingston Press. $1.25.

This little book consists to a large extent of a series of letters from well-known physicians who give the impression that they approve of the author's ideas concerning diaphragmatic respiration in the treatment of diseases of the heart, and tuberculosis. The author likewise gives a detailed description of the value of his window tent. In addition there are statements giving his ideas on tuberculosis and things in general which may or may not be of interest.

There is remarkably little of real value in this book.

Report of the Penrose Research Laboratory Formerly Laboratory and Museum of Comparative Pathology of the Zoölogical Society of Philadelphia. Herbert Fox. 30 pp. 1936.

This laboratory of comparative pathology, obtaining its material from the garden of the Philadelphia Zoölogical Society, is continuing its excellent work in advancing our knowledge. The continued interest of Dr. Fox and Dr. Ratcliffe has done a great deal to further the development of our knowledge of pathologic changes in wild animals. Such diverse findings are noted as nephritic changes in a Surinam toad, and gall stones in wild boar.

The Harvey Lectures. Delivered under the Auspices of the Harvey Society of New York. 1934-1935. William B. Castle, et al. Series XXX. 270 pp. Baltimore: The Williams & Wilkins Company.

Eight authorities, two of them foreign guests, contributed to this thirtieth series of Harvey Society lectures. Castle with his brilliant discussion of the etiology of macrocytic anemias, and Blake of Yale with his enthusiastic elucidation of collapse therapy in pneumonia, contribute most interesting and readable sections of a notable publication.

Diseases of the Respiratory Tract. Eighth Annual Graduate Fortnight of the New York Academy of Medicine. J. Burns Amberson, Jr., George Blumer, et al. 418 pp. Philadelphia and London: W. B. Saunders Company. $5.50.

The New York Academy of Medicine plans each year a graduate fortnight so that "the busy practitioner may be informed from authoritative sources as to the last word on a given topic. . . . Each year a subject is selected of outstanding importance in the practice of medicine and surgery; in the past years these have included disorders of the circulation, functional and nervous problems in medicine and surgery, tumors, disorders of metabolism and disease of the gastro-intestinal tract." Included in this fortnight are evening lectures, demonstrations,

and an exhibit at the Academy with co-ordinated clinics in important hospitals of the city in the afternoon. For the recent symposium the subject chosen was "Diseases of the Respiratory Tract", and the volume under discussion contains the evening lectures in this course.

The general subject is timely, and the selection of the sub-topics to be considered and the men to give the papers is excellent. Twelve of the twenty subjects are discussed by outstanding authorities in New York City, and eight men of special ability were called in from outside. The subjects and the speakers presenting them are as follows:

The Relation of Allergy to the Diseases of the Respiratory Tract, Maximilian A. Ramirez, New York City
Common Cold, A. R. Dochez, New York City
Sinus Disease from Infancy to Old Age, Charles T. Porter, Boston
Diseases of the Larynx, Trachea, and Main Bronchi, Charles J. Imperatori, New York City
Bronchoscopy in Relation to Diseases of the Respiratory Tract, Chevalier L. Jackson, Philadelphia
Bronchiectasis, J. Burns Amberson, Jr., New York City
Influenza of the Respiratory Tract, Henry T. Chickering, New York City
Chronic Pneumonitis, Jonathan C. Meakins, Montreal
Pneumonia in Childhood, Charles Hendee Smith, New York City
The Evolution of Pulmonary Tuberculosis, James Alexander Miller, New York City
Immunity in Tuberculosis, Arnold Rice Rich, Baltimore
Surgery of Tuberculosis of the Chest, Adrian V. S. Lambert, New York City
Pneumoconiosis with Particular Reference to Silicosis and Tuberculosis, Leroy U. Gardner, Saranac Lake
Emphysema, David Riesman, Philadelphia
Chronic Nontuberculous Empyema: Notes for the Physician and the General Surgeon, Howard Lilienthal, New York City
Abscess and Gangrene of the Lungs, H. Wessler, New York City
Pulmonary Thrombosis and Embolism, George Blumer, New Haven
Atelectasis, Massive Collapse, and Related Postoperative Conditions, Yandell Henderson, New Haven
Carcinoma of the Lung, Lloyd F. Craver, New York City
Diseases of the Mediastinum, Harrison S. Martland, New York City

Of particular interest to the reviewer were the lectures on the Common Cold and Immunity in Tuberculosis. He also felt that there was an especially good presentation of the facts relating to Influenza, the Evolution of Pulmonary Tuberculosis, Pneumoconiosis, Abscess and Gangrene, Thrombosis and Embolism, Atelectasis, Carcinoma, and Diseases of the Mediastinum. The subject of Emphysema deserves a more complete handling than is afforded in this volume.

On the whole the volume is of interest and importance to men devoting themselves largely to diseases of the lungs, and will also serve as it was intended that it should—to give the more general practitioner the important facts in the modern diagnosis and treatment of diseases of the respiratory tract.

Pathological Physiology and Clinical Description of the Anemias. William Bosworth Castle and George Richards Minot. Edited by Henry A. Christian. 205 pp. New York: Oxford University Press. $3.00.

One of the disadvantages of articles appearing in systems of medicine is that the information in them is usually available only to subscribers to the complete set. The Oxford University Press, however, has, of recent years, been in the habit of publishing certain important chapters in separate volumes. In the present instance, particularly, it must prove of great advantage to the practitioner, as this volume by Drs. Castle and Minot is unusually authoritative and complete. Not only well written, clear in expression and full of information available only in scattered papers in the recent literature, this book contains a most complete and accurate bibliography —a total of 646 references. The latter alone would make it a volume desirable to own, but the authors with a great deal of modesty say, "This bibliography is in no sense complete. Its objects are to suggest articles of interest to the clinician and to give access to a wider field of reading." As a summary of our present ideas in relation to the pathologic physiology of the anemias, there is no other volume at present available which in any way approaches this one in value.

Maladies de la Nutrition. F. Rathery. 173 pp. Paris: Masson et Cie, Éditeurs. 22 fr.

This volume, like the preceding ones of a series covering all the special phases of medicine, has been written primarily for the medical initiate. It is concerned with the diseases of nutrition and metabolism and especially emphasizes the disorders in those fields that have received the most attention and study during the past few years.

The writer, in the present book, follows the general scheme of the brochures that have preceded it. After dealing briefly with the normal digestive requirements and the normal fat, sugar and protein metabolism, the states which occur when the latter are pathologically altered are described in brief yet embracing fashion. The subjects of the minerals and the vitamines in their relation to the human economy are taken up in similar manner. Special emphasis is placed quite naturally on diabetes, gout and avitaminosis. The book as a whole is an ex-

cellent review of most of the accepted facts of the subject under consideration and although apparently rather brief, contains most of the essential details and more important information in the vast field of nutrition. It is highly recommended to the medical student and practitioner. It should be of value to the internist desiring a rapid review of the subject.

Medical History of Contraception. Norman E. Himes. 521 pp. Baltimore: The Williams & Wilkins Company. $7.00.

"The farther a notion reaches back into primitive times for its origin, the more universal must be its extent, and its power in history is rooted in this universality." (Lippert, "Kulturgeschichte".) "The desire for fertility control is neither time nor space bound. It is a universal characteristic of social life." Out of the misty nebula of legendary folklore, out of the cryptic references here and there in early historic literature, and from a detailed review of current publications, a firm-fisted scholar has grasped substantial, authentic material on which to build the story of attempts at fertility control among all peoples. After reviewing the practice among "preliterate societies" of Africa, America, and Australia, he tells how it was done by the Egyptians, the Jews, the Romans, and the Greeks.

The efforts in China, India, and Japan, to enjoy sex physiology without paying nature's price, and thus suffering famine or war or both, are outlined. The "History of the Condom or Sheath," which uses twenty pages, brings the story home to the occident, and brings the reader up to the "democratization of technique since 1800 in England and the United States". From then on through 200 pages it is a documented, detailed account of what we today call the "birth control movement" with its pros and cons, its ups and downs.

Perhaps because of his proved intimacy with his subject this capable author takes the liberty of expressing here and there, but particularly in the last two chapters on the "democratization of birth control" and on the "probable effects of democratized contraception," his sympathy with artificial control of positive fertility. He merits great credit that until these last chapters, which are mainly sociologic, he shows toward the practice and the mechanics of contraception a commendable scientific detachment, although, as he boldly exposes later, he is really a "tugwellian" materialist who rationally believes that a baby's worth may be expressed in economic and sociologic symbols. The enormous value of his book is not much diminished by this, for the philosophic and psychologic value of babies to the mates who must have them is a factor which almost all commentators on the subject miss, and, perhaps only the mates themselves can evaluate.

The reviewer's enthusiastic approval of this book could lead him astray into a discussion of these correlated values. He checks, only to add that this excellent history offers to each male and female who can think and act pertinently, plenty of information on a subject which profoundly affects the destiny of human beings. Professor Himes has erected a milestone in medical literature, marked to show where we have come from and the direction we are going in the control of fertility.

Experimental Studies on a Transmissible Myelomatosis (Reticulosis) in Mice. Otto Kaalund-Jorgensen. 142 pp. Copenhagen: Levin & Munksgaard. Swed. cr. 12:—

This detailed report summarizes nearly three years' work by the author in the field of leukoses in mice. It provides an interesting and stimulating companion work to the available studies by Furth on the lymphoblastic conditions in mice. The condition with which the author is working is of myeloid type and related to the transmissible leukoses of mice and guinea pigs, but essentially different from the transmissible fowl leukoses. The author regards the mammalian leukoses as being undoubtedly neoplastic in nature.

A Diabetic Manual. For Practitioners and Patients. Edward L. Bortz. 222 pp. Philadelphia: F. A. Davis Co.

This manual summarizes the fundamental knowledge of diabetes desirable for patients to possess. Special topics have been covered by contributing authors: Diet Therapy by Sister Maude Behrman; Diabetes in Children by Walter M. Bortz; Surgery by J. M. Deaver; Dental Care by C. F. Hellwege, D.D.S., and Care of the Feet by A. Rappaport, D.S.C.

The discussions of treatment, both of diabetes and its complications, the differential diagnoses of coma, and of diet planning are concise and simple. Nine types of diets, such as low-fat, low-protein, low-residue, high caloric and diets for different degrees of severity of diabetes are listed. Food tables include vitamin content and the composition of alcoholic beverages. The Sheftel urine testing outfit is recommended. This will prove a useful book for physician and patient, although the use of the new protamine insulin is barely mentioned.

Time of Ovulation in Women. A Study of the Fertile Period in the Menstrual Cycle. Carl G. Hartman. 226 pp. Baltimore: The Williams & Wilkins Company. $3.00.

Let no one who reads early in this book that "the male of Darwin's frog pipes his love call to the female only to receive the fertilized eggs stuffed down his vibrant throat, where he thenceforth incubates them in becoming, henpecked silence", or that the "2,000,000,000 eggs from which will arise the next generation of mankind would all fit into a derby hat," or that the "sperms to fertilize them could be encompassed by an ellipse the size of the capital 'O' of the print," be misled into underestimating this little book as the pot-boiler of a professional scribe. It is, on the contrary, a judicious scrutiny of all the useful research in animals and man which throws

light on the physiology of ovulation in woman; and a consideration of whether the knowledge we have thus acquired can be used in the control of human fertility.

With fine discrimination, the essential details of reproductive physiology are related as an introduction. What few scientific data there are on human menstruation is then sifted out of the mass of traditional assumptions and speculations. The author then settles down to consider what relation exists between the time of ovulation and menstruation. The scant and untrustworthy evidence which women themselves have volunteered is mentioned; that from the inaccurate assay of hormone changes is included and slightly overestimated; likewise that from the studies of hormone effects on uterine motility made by Knaus, and others, is given in detail and accepted with appropriate reservations. The evidence from the objective studies of ovaries by Ogino and others, especially by the author himself, is brought forward clearly and effectively; that from consideration of case histories of dated human coitus, productive or not, is properly discounted. The flimsy evidence from the finding of free eggs and the age of human embryos is not neglected. With becoming modesty, the author's own studies of the fertile period in the monkey, which throw the brightest light of all on to this obscure subject, are mentioned very briefly, but with expression of the fact that his are the only data which "are both comprehensive and conclusive". "There is an absolute Safe Period for the monkey-female." This justly famed biologist then indulgently includes a chapter on the "practical use of the rhythm of fertility and sterility", and with commendable conservatism opines "that most women would remain sterile by abstaining from sexual intercourse between the seventh and the twenty-first night after the beginning of the bleeding".

If the practical, clinical value of this excellent, timely discussion could be improved upon, it would be by the warning that an ovulatory menstruation, closely stimulating the shedding of a predeciduum, and unrecognized as a specious flow, is not uncommon in women over thirty-five, and is frequent in women over forty, and that when it occurs ripe follicles, ready to rupture any time, before, during, or after the flow, are usually present in the ovary. As pregnancy is possible when the follicle ruptures, for such women with such flow the safety of coitus cannot be even guessed at by regard for the so-called "menstrual" dates.

Orthopaedic Surgery. Walter Mercer. Second Edition. 906 pp. Baltimore: William Wood & Company. $10.00.

This most recent of textbooks on orthopedic surgery is written in a most clear and concise manner. At times brevity ceases to be a virtue, particularly in the discussion of therapy. The arrangement of the book is excellent. Subjects follow each other in logical sequence and the busy practitioner can find the information he desires with a minimum of effort. Tuberculosis of bones and joints is discussed in a most able manner. The prominence given to tuberculosis suggests that it is a far more serious orthopedic problem in Scotland than in America. On subjects where a difference of opinion can be debated, the data are carefully weighed and both sides are usually presented without prejudice.

The attitude of the book is one of conservatism, yet an open-minded conservatism. There is a well-chosen list of references to medical literature on each topic discussed. A few faults should be mentioned. In an attempt at completeness, particularly in the chapter upon "Affections of the Skeleton" a number of rare pathologic or roentgenologic variants are described which do not yet have the status of a disease entity. One would like to see a fuller mention of apparatus and physiotherapeutic procedures because of their great importance in the armamentarium of the orthopedic surgeon. The section on the back does not reach the clarity and completeness set by earlier chapters. The illustrations on the whole are well chosen, with a few exceptions.

The book reflects the English point of view on most subjects but shows familiarity with Continental European and American opinion as well. It must be adjudged the most readable and complete treatise on orthopedic surgery yet appearing in the English language.

International Clinics. Edited by Louis Hamman. Volume II. Forty-Sixth Series, 1936. 327 pp. Philadelphia, Montreal, London: J. B. Lippincott Company.

The rapid strides achieved in the basic sciences, physiology and biochemistry, insistently demand from the practicing physician studied effort to sustain his knowledge and interest in these fields. For upon them to a great extent does the clarity of conception of disease processes depend.

That the editor of this series is cognizant of this is attested to by the number of articles dealing with these subjects which in fact comprise the bulk of this volume. Thus we find discussed: The Erythrocyte Sedimentation Test, Pathological Physiology of Emphysema, The Syndrome of Hemoconcentration, Extracellular Body Fluid, and Nutritional Edema, among others.

It is more than a coincidence that these were written by men actually involved in bedside practice. Buttressed by other articles of interest, this volume should prove extremely valuable and interesting.

Medical Mycology. Fungous Diseases of Men and Other Mammals. Carroll William Dodge. 900 pp. St. Louis: The C. V. Mosby Company. $10.00.

In this book—the first of its kind in English—the author has accomplished the stupendous task of bringing together practically all the pathogenic

fungi known to man and animal. Morphology is discussed and keys to families, genera and species are given.

The first chapters are devoted to the general morphology of fungi, with definitions and occasional illustrations, the physiology of fungi including the influence of hydrogen ion concentration, the preparation of culture media, and methods of isolation of micro-organisms. One chapter is devoted to "The International Rules of Botanical Nomenclature." This nomenclature is followed throughout the volume.

The yeast-like organisms, their cultural characteristics, morphology, invasiveness and clinical appearance are discussed. The classification of many is of necessity based on heretofore published descriptions.

Many are inadequate as classification was based largely on fermentation reactions, the reliability of which is questioned. The author suggests further study on various types of media and finally on experimental animals to determine parasitism and pathogenicity.

In the chapters on Trichophytoneae—one group of the fungi imperfecti, the classification is based on that proposed by Sabouraud which attempts to correlate the morphologic and clinical characters. Here especially the author has introduced clinical description of various dermatoses with their etiology. Treatment of the more common type of fungus infections as ringworm and athlete's foot is included also. To many of these statements the dermatologist with mycologic experience will take exception.

Of necessity the subject matter is condensed, perhaps too much so, so that the beginner in medical mycology is baffled by the immensity of the subject. However, the well-arranged and complete index is most helpful in clarifying synonymous terminology.

One of the chief values of the book lies in its extremely comprehensive bibliography which makes it a source of practically all of the known mycologic literature. It is a most valuable adjunct to any mycologic library.

Handbook of Surgery. Eric C. Mekie. 699 pp. Baltimore: William Wood & Company. $4.50.

It is stated in the preface of this admirable handbook that its purpose is to prepare the undergraduate student for the passing of his final examination in surgery at the Royal Infirmary in Edinburgh and that "all that is now essential to the task of surmounting the final hurdle of the medical undergraduate career is excluded." After careful reading, the reviewer heartily agrees with John Fraser in his foreword that the "author has covered the field so thoroughly that the *postgraduate* (and such a term has a wide inclusion) will find much to interest him."

It is a handbook of convenient size bound in flexible cloth covers containing about 700 pages of excellent typography including a good index of 14 pages. The text is presented throughout with topical and subtopical headings and information of differential nature is often presented in tabular form. The supplemental 24 figures are well chosen, are diagrammatic, and remarkably purposeful. The text is divided into 41 chapters. In addition to the above noted features, which appeal to the reviewer, are the introductions to the chapters. These introductions in most instances present tersely the anatomy, embryology, physiology and pathology of the organ or anatomic region to be considered. To the reviewer an understudy of such is fundamental in the teaching of surgery. Without such a foundation upon which to build, he believes the teaching of surgery lacks sound practical value to the student. The teaching of the technic of various operations without such fundamental knowledge is like a heavy structure built on shifting sand. A good surgeon should not only possess technical skill (which is largely inherent and cannot be but partially acquired by teaching) but he should have sound knowledge of anatomy and morbid anatomy. He should understand physiology and should be able to recognize anomalies quickly. The reviewer believes that this volume is not only a sound, practical handbook (designed, as is stated in the preface, for the undergraduate in surmounting his final examination) but one of immense practical value to the young surgeon in practice. He will not find in it any description of the technic of operations but he will find in it information fundamental to the practice of intelligent surgery.

Physician, Pastor and Patient. George W. Jacoby. 390 pp. New York and London: Paul B. Hoeber, Inc. $3.50.

Jacoby out of a rich and unusually fruitful experience of many men of many minds, with a colorful background of history, traces the relationship from the earliest days of religion and medicine. He deals judiciously with many of the moot topics to which men are sincerely giving thought; euthanasia, vivisection, birth control, the divorce problem, and the co-operation of clergyman and physician for mutual help to the patient. Beginning, as much of it did, in superstition and empiricism, medicine has had to fight its way, touched at many points by Judaism, Mohammedanism and Christianity. There is still a place where the liberal priest and open minded physician can meet to the advantage of us all, in the management of mental diseases, in legislation, and in education of the public.

The author is to be congratulated on his masterly management of an extremely difficult subject. Many a patient and pastor, as well as his doctor, will find much food for thought in the pages of a book that is decidedly a credit to both author and publisher.